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FOUNDED BY JAMES PLEASANT PARKER

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LIII.

TREATMENT OF OTITIC LEPTOMENINGITIS.*

PLEA FOR INVESTIGATIONS IN TWO DIRECTIONS: (1) KUBIE'S
THEORY OF FORCED DRAINAGE; AND (2) DIRECT
SURGICAL DRAINAGE.

PHILIP D. KERRISON, M. D.,

NEW YORK.

In spite of the immense amount of animal experimentation and surgical effort by distinguished workers in all civilized countries, it will not be denied that the surgical treatment of otitic leptomeningitis has yielded only meager results. We have no rational, clearly defined and generally accepted plan of surgical treatment which in any large series of cases can be depended upon to save even a small percentage of lives.

So long ago as 1913, Dr. E. W. Day¹ of Pittsburgh published a report of 53 cases of otitic meningitis operated upon in his hospital practice with autopsy reports in 38. The surgical measures employed included (a) dural incisions; (b) drainage of the lateral ventricles; (c) through and through irrigation from the

*Delivered as a special presentation before the American Otological Society, Atlantic City, May 17, 1932.

lateral ventricle to a needle in the lumbar canal; and (d) drainage through the cisterna magna. Day's conclusions were succinctly stated as follows: "Theoretically, surgery should give relief; practically the mortality has not been changed by it." This unbiased report by a surgeon of recognized standing and skill is particularly interesting when we reflect how little, if any, has been the practical surgical advance since it was written.

In more recent years, Dr. W. E. Eagleton has experimented with the following procedures: (a) subarachnoid irrigation from a point over the frontal lobe to the cisterna magna;² (b) subarachnoid irrigation with replacement of fluid withdrawn by Ringer's fluid;³ and (c) ligation of the homolateral common carotid artery.⁴

Withdrawal of Fluid, Either Cerebrospinal or Spinal, With Replacement of Ringer's Fluid.—The rationale of this procedure is not clear. To expect to influence a meningeal infection by withdrawal of a limited amount of fluid from the cisterna magna or spinal canal would seem hardly more logical than would be an attempt to control a sinus infection with bacteremia by the simple withdrawal of blood from the homolateral jugular vein; for in either case the activating focus of infection would remain operative. As to the replacement measure, it is difficult to conceive how the introduction of a like amount of Ringer's fluid could in the slightest degree neutralize the infection or in any way promote recovery.

Ligation of the Common Carotid Artery.—The theory that ligation of the common carotid will promote the processes of repair in otitic meningitis by reducing arterial pulsations and thereby placing the inflamed meninges at rest is not likely to meet with general acceptance by surgeons or physiologists until more convincing evidence is brought to its support than has yet been adduced. Surgical literature affords a rather formidable list of seriously untoward results following carotid ligation, and until animal experimentation by trained and competent workers can establish a scientific justification, so drastic a measure must bear the stigma of empiricism and must therefore withstand the opposition of many conservative surgeons. But aside from the general considerations above noted, it would seem to me that there are

abundant physiologic grounds, which I have not the time to elaborate here, for the belief that, rather more than other organs of the body, the subarachnoid system is a vital mechanism of which it is particularly important, in health and rather more so in disease, that the arterial supply should not be interfered with.

To repeat: We are still without any recognized surgical method upon which a reduction of mortality can be predicated.

The purpose of this paper is to urge the importance at this time of investigations in two directions: i. e., (1) of the theory of forced drainage; and (2) of direct surgical drainage.

Kubie's Theory of Forced Drainage.—I do not know how generally this important therapeutic hypothesis has engaged the attention and interest of the members of this society. Its possibilities, however, are so obvious and interesting that I shall try to outline the theory on which it is based.

According to modern theory, the cerebrospinal fluid is a dialysate—i. e., a filtrate from the capillaries of the choroid plexuses and in smaller amount from the capillaries of the perineural and perivascular spaces of the central nervous system. This, at least, is the view to which a great majority of the later investigators strongly lean. This question is not merely of academic interest but has a practical bearing on modern therapeutic theory. It has been ably discussed in a paper by Fremont-Smith of Boston.⁵

The cerebrospinal fluid is normally in osmotic equilibrium with the blood. If the blood is diluted, as by the intravenous injection of distilled water or of any hypotonic solution or by the forced drinking of large amounts of water, the osmotic equilibrium is disturbed and there quickly follows marked and progressive increase of intracranial pressure and of the production and flow of cerebrospinal fluid. On the other hand, the intravenous injection of a hypertonic solution—i. e., of a solution sufficiently loaded with salts to bring the osmotic pressure of the blood above that of the cerebrospinal fluid, is followed by the opposite train of results—i. e., reduced intracranial pressure, some temporary reduction of brain bulk and diminished production and flow of cerebrospinal fluid. In other words, intracranial pressure and the flow of cerebrospinal fluid may be changed at will and in either direction by dilution or increased concentration of the blood. In

the above I have merely stated facts which have been proved and corroborated by Weed, Kubie and many other investigators of recognized scientific standing. In part these facts are not new, having been solidly built upon experimental investigations extending back over the past fifteen years.

In 1923,⁶ Dr. Lewis H. Weed carried out an extensive series of technically exact experiments with the purpose of checking or supporting the thesis that the choroid plexuses are chiefly responsible for the production of the cerebrospinal fluid. These experiments were upon anesthetized dogs and dealt solely with the effects of intravenous injections of distilled water and of various hypotonic solutions. Leaving out of our consideration the exceedingly interesting pressure variations noted in the cisterna magna, the sagittal sinus and in the arterial blood entering the brain, the fact which chiefly concerns us is the great and progressive increase in intracranial pressure which regularly followed this method of reducing the osmotic value of the blood. Following these experiments, the dogs were immediately killed for post-mortem examination, which in every case showed extensive morphologic changes in the cells of the choroid plexuses and, extraventricularly, in the cells of the perineural and perivascular spaces of the central nervous system. An interesting and significant fact, later corroborated by Kubie, was the considerable distension of the perineural spaces everywhere noted in spite of the enormous increase of intracranial pressure, an increase of pressure in some cases so great that the cortical convolutions were found flattened against the inner wall of the skull. This interesting physiologic phenomenon should be borne in mind for its bearing upon the theory which I shall try to outline.

Weed's conclusions were that his experiments supported the theory that the choroid plexuses are responsible for the great bulk of the cerebrospinal fluid, but that a certain infinitely smaller portion is derived from the capillaries of the perineural and pericellular spaces of the central nervous system, finding its way by perivascular pathways to the subarachnoid space. The extraventricular portion of the cerebral fluid, though in relatively small ratio to the total bulk, plays an important part in Kubie's theory.

Dr. Lawrence S. Kubie,⁷ I believe, was the first to see the

therapeutic possibilities of Weed's experiments if combined with continuous drainage from the cisterna magna or lumbar canal (Fremont-Smith). His experiments also were upon dogs and were carried out with minute attention to technical detail. His scheme called for continuous drainage from the cisterna magna and pressure observations through a trephine hole in the parietal bone. The dura presenting in the parietal opening thus provided was incised with care not to enter the subarachnoid space. Into the circular trephine hole a specially constructed brass cannula was screwed, and the joint thus formed was further sealed with dental cement. The cannula was filled with white oil which was carried through a series of connections to a manometer which registered all variations of intracranial pressure.

The occipito-atlantoid ligament was next exposed surgically and through this the cisterna magna was entered by means of a 16-gauge lumbar puncture needle. The needle was then connected with a second manometer by means of a metal three-way adaptor. By this mechanism Kubie was able, by the simple turning of a stop-cock, either to drain the cisterna or, by closing it, to record the pressure variations within the cistern when the outflow from the cisterna was shut off.

By this seemingly complicated but practical apparatus he was able, by means of the parietal manometer, to observe and record the pressure changes, varying from 2 to 5 mm., with each heart-beat, the much greater variations following inspiration and expiration, the pressure reductions when outflow from the cisterna was established and finally the pressure changes following intravenous injections of hypotonic solutions. •

In kind, the reactions and records of Kubie's experiments corroborated those of Weed. The effect of intravenous injections of hypotonic solutions was an increase of intracranial pressure and an increased flow of cerebrospinal fluid. Naturally, with continuous drainage from the cisterna, the increase of pressure and its variations were relatively small.

Dr. Frank Fremont-Smith, working in collaboration with Dr. Tracy J. Putnam and Dr. Stanley Cobb,⁸ has further elaborated Kubie's method, substituting lumbar puncture for cisternal drain-

age, and the forced ingestion of large amounts of water by mouth—i. e., 500 to 1,000 cc. per hour, for intravenous injections. Water in such amounts inevitably produces diuresis which would naturally interfere with the desired dilution of the blood. To forestall this result, a subcutaneous injection of 1.0 cc. of posterior lobe pituitary extract is given. This delays diuresis from four to six hours, during which "renal lag" the drinking of large amounts of water should produce marked dilution of the blood. Apparently this theory has been substantiated.

As a resumé of the histologico-pathologic course of events under this treatment, the following quotation from a paper by Kubie is both interesting and instructive. Speaking of an induced meningismus or meningitis, he says: "(1) Early in the acute stage of a meningeal irritation and throughout all later stages, the perivascular spaces and many minute crevices of the central nervous system become lined with small mononuclear cells which we believe to be lymphocytes. (2) Prolonged drainage of the subarachnoid space during the meningeal reaction is accompanied by a gradual alteration of the cells of the cerebrospinal fluid; the lymphocytes increase in relative abundance and finally . . . far outnumber the other elements. (3) Intravenous injections of hypotonic solutions which are begun when the spontaneous flow of fluid has almost ceased are accompanied by a renewed flow of fluid and a still greater increase in the percentage of lymphocytes. (4) Sections of the nervous systems of animals treated in this way show that the perivascular plugs of lymphocytes are largely extruded into the subarachnoid space and must be the source of the increasing proportion of these cells."

As to the risks and ultimate results of the procedure, Kubie makes the following pertinent statement: "That the procedure is quite innocuous, at least to normal animals, is shown by the fact that many dogs were subjected to this treatment repeatedly at intervals of only a few weeks without any injurious effects."

Twenty or twenty-five years ago the glittering possibilities of subarachnoid irrigation held the minds of students of meningitis and continued to sway their thoughts until 1919, when the published investigations of Weed and Wegeforth⁹ proved to thinking minds its risks and practical futility. But even had subarachnoid

irrigation been mechanically feasible and devoid of its inherent risks, it seems clear, in the light of present knowledge, that its effect might have been simply to wash out the larger spaces of the subarachnoid system, leaving untouched the cell-filled and germ-infested perineural and perivascular crevices of the central nervous system. Compare with this the theory on which Kubie's hypothesis is based: a lavage of the subarachnoid system having its initial movement in the perineural crypts and crevices of the nervous system; a drainage system in which its own accelerated fluid movement plays the leading part.

Personally, I am immensely impressed with the ultimate constructive value of Kubie's theory. As to application of his technical method to human meningitis patients, that, of course, is another question. The human brain in proportion to body bulk is more complex and certainly larger than in any other animal. With a lesion like meningitis, where intracranial pressure is already greatly increased, the risks from any procedure raising pressure still further would therefore be proportionately greater. Even in his experiments upon dogs, however, Kubie allows a period of drainage through the cisternal needle, before giving the intravenous injection of water, which precaution would of course be applicable to the meningitis patient. To me this theory, which is still under investigation, seems the most interesting and important development of recent decades.

Resumé of Facts and Hypotheses Upon Which Modern Theory Is Based.—(1) Strong drift of opinion to the effect that the cerebrospinal fluid is a dialysate or filtrate from the capillaries, rather than a glandular secretion (Weed, Fremont-Smith);

(2) The cerebrospinal fluid is in osmotic equilibrium with the blood, and its production and flow may be increased or diminished at will by dilution or increased concentration of the blood (Weed);

(3) Following intravenous injections of hypotonic solutions, the perineural and perivascular spaces are markedly distended in spite of enormous increase in intracranial pressure (Weed);

(4) In inflammatory conditions of the central nervous system and of the meninges, the perineural and perivascular spaces are

lined with newly formed cells, many of which are lymphocytes. Following intravenous injections of hypotonic solutions accompanied by continuous drainage of the cerebrospinal fluid, masses of these cells may be seen at postmortem to have been extruded into the subarachnoid space (Kubie);

(5) Dogs upon which these experiments have been repeatedly made and at intervals of only a few weeks have made apparently perfect and permanent recovery (Kubie).

The theory, then, is that the perineural and perivascular spaces, which during meningeal inflammation are the supply posts for the enormous increase in the number of lymphocytes, are also the storage points of pathogenic micro-organisms; and that since these cell-filled and infected crevices are in part the normal starting points of fluid production or dialysis, a stimulated increase in fluid production may rid the central nervous system of toxic matter through an autogenous cleansing of its principal storage points.

Before leaving this subject, let me put myself straight as to what seems to be its present therapeutic status. It has been experimented with in multiple sclerosis, anterior poliomyelitis and to a limited extent in purulent meningitis, and in none of these diseases has its therapeutic value been definitely established. In the main the men who have conducted this investigation are not of the type of mortals whom enthusiasm seizes by the throat and prematurely runs away with. So far as I have been able to learn, the results in multiple sclerosis have been nil; in anterior poliomyelitis they have been promising; in meningitis, I know through private but reliable sources of one case, not of otitic origin, which recovered after five weeks of almost continuous spinal drainage. The facts which impress me are (a) the extremely logical basis of the theory itself; and (b) Kubie's demonstration that the procedure itself is not dangerous to healthy dogs, and the theoretic presumption that it would not be to healthy men.

As a body, aural surgeons probably see more cases of purulent leptomeningitis in the course of a year than any other medical group. The question I have been asking myself is whether otologic surgeons, with their composite theoretic and technical knowledge, might not utilize the facts as I have tried to present them.

to the good of humanity. I believe that this society should interest itself in this investigation. It would be fortunate indeed if it should attract what might be termed a selected group or groups within its membership. By "selected," I mean men of scientific and technical bent and ability, working in well equipped university or institutional laboratories, rather than in general hospitals.

Surgical Drainage.—In venturing to speak of surgical drainage I want to make one point clear. I am not presuming to advise or suggest to the members of this society a surgical method to be applied to the treatment of sufferers from otitic meningitis. Rather I am suggesting a line of investigation which might lead to conclusions upon which a modification of surgical theory or method might be formulated. In describing a surgical procedure in detail, therefore, I am adopting that method merely as the easiest and clearest way of indicating a series of animal experiments which might or might not lead to a practical surgical advance.

Anatomy.—Rather loosely speaking, the ventricular and sub-arachnoid systems perform two mechanically separate and distinct functions. In the lateral, third and fourth ventricles, the great bulk of the cerebrospinal fluid is formed. In the sub-arachnoid system of communicating spaces the ventricular fluid is distributed to the cortical surfaces of the brain and ultimately to contact with the large venous sinuses through which it is converted and absorbed into the venous blood stream. The so-called "cerebrospinal circulation" implies therefore little more than the course which the ventricular fluid must travel from its points of origin—i. e., from the lateral ventricles through the foramina of Monroe to the third ventricle; thence through the aqueduct of Sylvius to the fourth ventricle; and from the fourth ventricle through the foramen of Majendie and foramina of Luschka into the cisterna magna. This is the first half of the journey. The extraventricular flow has been made the subject of intensive study. From the cisterna magna and presumably from the basal cisterns the fluid movement is not that of fluid seeking a lower level but rather may be compared to a spreading fountain with the central projection vertically upward toward the great longitudinal sinus and a lateral spread to the cerebral cortices and the

lesser venous sinuses—particularly toward the lateral and sigmoid sinuses. This general scheme of distribution is obvious because physiologically essential and is of practical significance in any logical topographical theory of surgical drainage.

The Cisterna Magna (Cisterna Cerebellomedullaris).—Because of its large size and easy demonstration on the cadaver, the cisterna magna is the one best known to surgeons generally and perhaps also to students of otology. As a matter of fact, in the pathology of otitic meningitis, it is probably of all the cisterns the least important, its chief and paramount significance being the fact that it constitutes the great basal reservoir into which the ventricular fluid is first discharged.

The Pia-arachnoid Cisterns.—We clarify our view of these cisterns if we remember that such a space exists wherever the pia leaves the arachnoid to invest the structures forming a depression, the arachnoid bridging the cavity so formed. We know also that in certain regions pia-arachnoid separations occur as much in response to the need of a protective water-bed as to the conformation of adjacent structures. Two such regions, or spaces, to which I shall next refer, might perhaps be kept in mind with advantage to surgical thought and clarity.

The Tentorial Spaces.—If we examine a skull from which the brain has been removed with minimum disturbance of dural linings and partitions, we shall find that the tentorium is a strong, tense structure, smooth alike on its upper, or cerebral, and its inferior or cerebellar surface. If, therefore, the pia invests the uneven upper surface of the cerebellum with its slight depressions between vermis and hemispheres, and shallow linear grooves between laminae and lobes, we must assume that a system of communicating spaces exists between it and the taut surface of the tentorium above. Similarly, a space must be assumed between the still more uneven inferior surface of the brain and the upper surface of the tentorium. Furthermore, we must remember that the tentorium supports the entire weight of the occipital and of very considerable portions of the parietal and temporal lobes. Unless, therefore, we discard the accepted water-bed theory of its function—i. e., that the cerebral fluid is so distributed as to guard the various parts of the brain from shock and injury, it seems

necessary to assume the presence of cerebrospinal fluid in adequate and very considerable amount on both sides of the tentorium. If we wish to give names to these water-bed spaces, they might well be called the cerebrotentorial and the cerebellotentorial basins, respectively.

Pathology.—Owing to the limited time at my disposal, I shall make only passing reference to the pathology of the disease. Naturally, I have read many postmortem reports. For the purpose of practical surgery many of them, while interesting and instructive, are not sufficiently clear or definite in indicating the regions or structures chiefly involved. From the correlated statements in many autopsy reports one receives the impression that, of the various subarachnoid cisterns described in textbooks, those most frequently involved in otitic meningitis are the following: the cisterna interpeduncularis, the cisterna chiasmatis and the cisterna fissuræ Sylvii (Tilney and Riley). In other words, from their frequent and extensive involvement, it seems probable that infection of these spaces must exert no small influence upon the spread of a basal infection and therefore upon the appalling mortality of the disease. Possibly next in pathologic importance are the cisterna pontis and the superior cerebellar cistern.

Of all the pathologic reports which I can recall, I know of none more suggestive in its surgical implications than one with which all of you are probably more or less familiar. I refer to the paper which Dr. E. D. D. Davis read before the British Royal Medical Society in 1921.¹⁰ This report was based on the autopsy findings in twelve cases of otitic meningitis. In it he makes five statements to which I ask your particular attention on account of their bearing not only on the pathology of the disease but also on the theoretic lines of surgical treatment. He states (1) that in each and all of the twelve autopsies large collections of pus were present between the tentorium and the cerebellum; (2) that pus was invariably found in the cisterna interpeduncularis; (3) that in these cases the optic chiasma was surrounded by pus. This, of course, was equivalent to saying that the cisterna chiasmatis contained pus; (4) that in the pathologically more advanced cases meningitis was beginning to spread along the Sylvian fissure to the cerebral cortex; and (5) that "the cisterna

magna in some cases was free of pus and that collections of pus in this region was not a marked feature in any case." These statements are the more convincing in that they are so absolutely logical. "The cisterna interpeduncularis," says Gray, "extends forward over the optic chiasma, forming the cisterna chiasmatis." When one, therefore, is extensively involved in a suppurative process, so presumably will the other be. Just external to the cisterna chiasmatis and partly separated from it by the optic nerve and olfactory trigone is a considerable depression which marks the beginning of the stem of the fissure of Sylvius. This depression, which is bridged over by the arachnoid in its passage from the temporal to the frontal lobe, is called by Tilney and Riley the cistern of the fissure of Sylvius (*Cisterna fossæ cerebri lateralis* of Gray).

The point I am trying to emphasize is the fact that these cisterns are directly intercommunicating. For descriptive purposes their separation under different names has probably served a useful purpose. Surgically, this division has probably conduced to confusion of thought by making the problem of surgical treatment seem difficult beyond human possibility of accomplishment. In other words, in relation to a basal meningitis, we might have a better conception of their significance if we recognized these three classical cisterns as constituting one great central basin of infection. If we could drain one, it is possible that we might drain all three. (Fig. 1.)

Cerebrospinal Stasis.—Before leaving the subject of this important pathologic center, I should like to refer briefly to a factor in the mechanics of an otitis meningitis which seems to me to have escaped proper emphasis—i. e., cerebrospinal stasis. This condition is perhaps best brought out by certain differences between infections of the vascular and of the subarachnoid systems. In a lateral sinus infection with bacteremia, for example, we have a bacteria-laden blood stream which, until recovery occurs or death supervenes, will continue to circulate with relatively normal regularity and rhythm. Not so with an otitic leptomeningitis. As soon as a subarachnoid infection has progressed to a certain undetermined point, we are led to believe that nature's first response is an increased production of cerebrospinal fluid. This

undoubtedly is a conservative process, tending to sweep septic matter from the earliest focus or foci of infection into the main channels by which it is conveyed in diluted form to the venous blood stream. If protective barriers are not formed the pathologic picture quickly changes. Intracranial pressure rises quickly. Lymphocytes and leucocytes are produced in ever increasing numbers—lining and crowding the perineural and perivascular spaces. Cerebrospinal fluid is poured from the ventricles in quantities progressively greater than its possible absorption into the venous circulation. Sooner or later it accumulates in certain basal cisterns in such quantities and the outlets therefrom are so obstructed by peripheral pressure that circulation is in practical abeyance. In other words, there is developed a condition of relative or practical stasis, in the presence of which spontaneous resolution or recovery would seem practically impossible. Theoretically any cortical incision providing an escape of cerebral fluid should initiate a movement relieving this situation. Practically we know that the problem is not so simple.

That a single cortical outlet for the escape of infected cerebral fluid, however perfect its initial results, is not sufficient to turn the scale in a diffuse otitic leptomeningitis would seem to be proved by Dr. John D. Richard's unpublished experiences with drainage through the labyrinth after removal of the modiolus. Through Dr. Richard's courtesy, I saw with him some years ago, not in consultation, two cases in which he had established what seemed to be perfect drainage by this route. In these cases he was able, by means of a small curette, periodically to re-establish drainage—with welling up of cerebral fluid into the tympanic cup and with great temporary relief of symptoms. Both patients ultimately died of meningitis which, he told me, had up to that time been true of all such patients under his care. If my memory serves me, his theory then was that ultimate failure was due to the gradual formation of adhesions around the internal auditory meatus which finally could not be reached or corrected by a labyrinthine curette. Another theory which has occurred to me is this: that drainage through the labyrinthine or modiolar route, as it probably would be from any single cortical outlet, was not sufficient in amount to relieve adequately the cerebrospinal stasis

in the basal cisterns or other spaces which finally became blocked by surrounding adhesions or by pus agglutinations to a degree relieving pressure upon the internal auditory meatus.

In almost every surgical discussion, dural incisions are mentioned as a surgical measure to be considered. In a half-hearted and haphazard way they have been extensively employed. So far as I know, however, no studied plan has been evolved or attempted to bring the incisions into anatomic relation with the gross pathology of the disease. It would seem, therefore, that any topographical scheme having such ends in view should, even though based on purely theoretic grounds, be worthy of consideration.

That surgical drainage in otitic meningitis has never been fairly tested is indicated by the fact that in surgical textbooks and monographs little or no attention has been given to the preparation of the surgical field which for this, more perhaps than for any other major operation, is an absolutely essential preliminary step. In fact, a careful study of the prepared surgical field is not only a spur to interesting and perhaps constructive speculative thought, but is also essential to a full appreciation of certain physiologic factors which should or might aid the surgeon in his combat with the disease. If in the following paragraphs I describe this preliminary work in the somewhat dogmatic method usually employed for an approved and accepted operation, I hope it will be understood that I adopt this method solely in the interest of brevity.

Roughly speaking, a dural exposure adequate for a combined operation for infective sinus thrombosis and for the exploration of a hypothetic temporosphenoidal abscess would provide the essential area through which to drain a diffuse purulent leptomeningitis of otitic origin. Over the posterior cranial fossa the first guide to the removal of bone would be the line, with which we are so familiar, followed by the lateral sinus, considering the horizontal and the descending, or sigmoid, portions as one vessel. The sinus should be uncovered from a point roughly marking the junction of the middle and torcular thirds of the horizontal portion and extended forward and downward to the disappearance of the sigmoid portion in the jugular foramen. Throughout the length

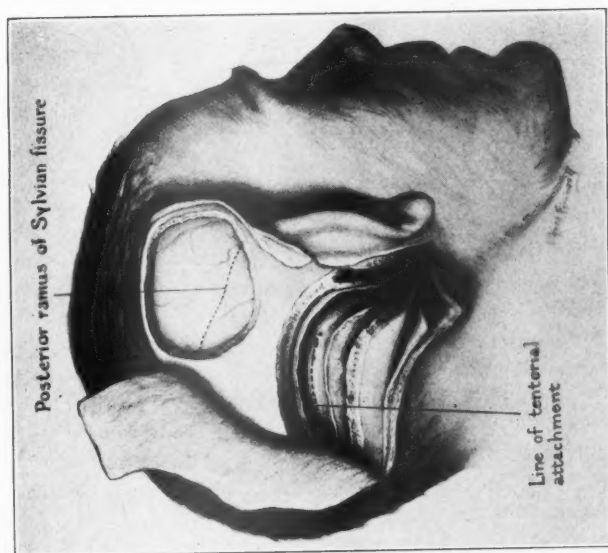


Fig. 2.



Fig. 1.

of this exposure the inner wall of the sinus constitutes the logical area for important incisions. In fact, throughout the exposed part of the horizontal portion the inner sinus wall provides the logical and only practical area through which to reach and drain the two tentorial basins. They cannot be adequately drained through incisions located elsewhere. Ligation of the jugular vein and obliteration of the sinus and removal of the outer sinus wall constitute therefore essential preliminary steps. In a general way, this part of the operation is identical with the classical procedure for infective sinus phlebitis, with the difference that rather more bone on both sides of the sinus* should be removed. When this work is completed, we have accomplished the following results: (1) We have practically eliminated the risk of serious venous hemorrhage; (2) by means of the compress at the torcular end of the lateral sinus and consequent retardation of venous return flow, we have presumably increased arterial tension in the ventricles and thereby increased the production of cerebrospinal fluid; and (3) we have provided the ideal drainage area from regions the frequent involvement of which in otitic meningitis has been attested by innumerable autopsy reports.

The inner triangular plate of bone in front of the obliterated sigmoid sinus should now be removed, the bone removal being carried sufficiently forward to permit of satisfactory inspection of the dura in this region. But unless there is special reason or indication therefor, loss of time in making a very extensive exposure in the forward direction of the internal auditory meatus would seem to me to be generally uncalled for.

Bone must next be removed over the tegmen tympani et antri, exposing the temporal lobe much as for exploring a temporo-sphenoidal abscess. This dural exposure will again be referred to in relation to drainage of the midcranial fossa.

This preliminary operation for essential dural exposure may seem a formidable tax on the patient's power of resistance but is probably less so than the shock of an attempted cerebrospinal irrigation. It may be necessary, however, and would certainly be wise, to have provisions for a blood transfusion before, during or immediately following the operation. We may now consider

this prepared surgical field separately in its different parts and in brief detail.

Posterior Fossa Region.—To appreciate the logic or necessity of obliteration of the lateral sinus or, speaking more exactly, of obtaining access to its inner wall, we must bear in mind that the lateral attachment of the tentorium to the sides of the skull is through the medium of the inner wall of the horizontal portion of the lateral sinus to the median line of which it is directly affixed. Two series of vertical incisions, therefore, one above and the other below the attachment of the tentorium, should provide the logically best drainage outlet from the two tentorial basins.

Surgical Value of Intracranial Pressure.—So closely associated with the possibilities of surgical drainage is the flow of cerebrospinal fluid that I would like to say a word as to its quantitative variations in health and in disease. That we know little of the average normal amount, even within physiologic limits, is indicated by the divergent statements of recognized authorities. Thus, Howell¹¹ estimates it as varying from 60 to 80 cc., while Tilney and Riley¹² place the normal variation at from 100 to 150 cc. The important consideration, however, is the fact that the amount regularly undergoes great increase in the presence of any irritative or inflammatory lesion of the meninges. Thus Howell¹³ states that "in fractures of the base of the skull the fluid has been observed to drain off at the rate of 200 cc. or more per day." The importance of this fact in relation to surgical drainage is obvious. In other words, it is clear that if ever we are able to devise a practical system of drainage pathways from the principal foci of infection, increased flow of cerebral fluid may be confidently counted upon as a powerful factor in ridding the brain and subarachnoid system of septic matter.

I beg you now to consider with me for a few moments the normal flow of cerebral fluid in health in relation to this surgical field. In so perfect a mechanism as the subarachnoid system nothing, we may assume, is without ordered purpose and significance. We know that after its expulsion from the ventricles, the flow of cerebral fluid is toward the large venous sinuses of the brain. In the anatomic adjustment to this physiologic purpose and ne-

cessity there must, therefore, be natural and probably permanent pathways from the great subarachnoid cisterns at the base of the brain toward the larger sinuses.

Excluding the great longitudinal sinus, the lateral sinus constitutes the second great venous reservoir toward which the cerebral fluid absolutely must flow. If, therefore, after obliteration of the sinus, we provide the two series of vertical incisions previously referred to, we shall have created two series of dural outlets toward which physiologic necessity has presumably provided natural sluiceways.

With regard to the region of the obliterated sigmoid sinus: this part of the sinus has, of course, no connection with the tentorium. It does, however, mark a continuation of the regional line toward which the cerebral fluid normally flows. Two or three incisions, therefore, following the course of the sinus and at right angles to its long axis should drain this region and possibly at the same time provide an indirect drainage pathway from the cisterna pontis.

The Temporosphenoidal Field presents greater theoretic and technical difficulties than that of the posterior fossa. In the first place, the brain in its dural capsule provides in itself no recognizable surgical landmarks, the position of subdural structures being determinable only by their known relation to bony points in the skull. (Fig. 2.)

One cannot look upon this prepared surgical field without thinking of the great central basin composed of the cisterna interpeduncularis, the cisterna chiasmatis and that of the Sylvian fissure. The cisterna interpeduncularis itself might be reached by a straight incision or puncture from the outer cortical surface of the temporal lobe, and the approximate cortical starting point for such an incision would not be difficult to locate. But when we consider the associated or surrounding vessels—e. g., the intracranial trunk of the internal carotid, the cavernous sinus, and particularly the thick plexus of small veins entering the sinus—the risks of a practically uncontrollable hemorrhage are so great as to be deterrent. On the other hand, it may be assumed that the pia and arachnoid investing the inferior surface of the temporal

lobe are in communication with the pia-arachnoid investitures of the cisterna interpeduncularis. Theoretically, therefore, an incision starting from a point external to the cistern and incising the inferior surface of the temporal lobe in a direction from within outward might provide a drainage pathway for the escape of cisternal fluid. Such an incision, however, would inevitably sever the anterior branch of the middle meningeal artery and the traumatism incident to the control of hemorrhage might more than counterbalance any therapeutic advantage. This brief discussion is simply to indicate the difficulties and dangers of attempted direct drainage by this route, the value of which at best would be more than doubtful. Even were there no surgical risks, it would be excluded as contradictory to the theory on which this topographical scheme is based—i. e., the provision of cortical outlets in regions toward which the normal flow of cerebral fluid is physiologically directed.

The Fissure of Sylvius differs from the other great cerebral fissures in its relatively early development. It is apparently formed by the folding together of different parts of the early primitive brain rather than as a space between cerebral lobes of subsequent development as is the case with other cerebral fissures. It is present, according to Gray, as early as the third month of fetal life, about three months before any of the other great fissures are demonstrable. In the fifth fetal month it exists as a great cleft or fossa, while the brain cortex still presents a perfectly smooth surface with no indication of sulci or convolutions. (Fig. 3.) That it is a common pathway in postnatal life for the spread of a meningeal infection is attested by many autopsy reports. Its direct communication through the cistern of the same name with other basal basins is anatomically demonstrable. A series of incisions through and at right angles to the posterior arm, or ramus, of the Sylvian fissure should therefore provide drainage outlets from the midcranial fossa and from the central basal cisterns.

The Posterior Ramus of the Sylvian Fissure.—It is not easy to make any statement as to the surgical position of this cleft in the outer cortical surface of the hemisphere which will carry conviction. As said before, the dural covering of the brain in itself provides absolutely no landmarks. Furthermore, even were it

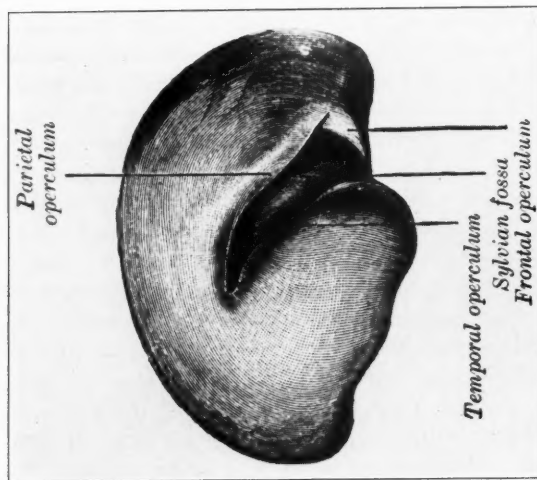
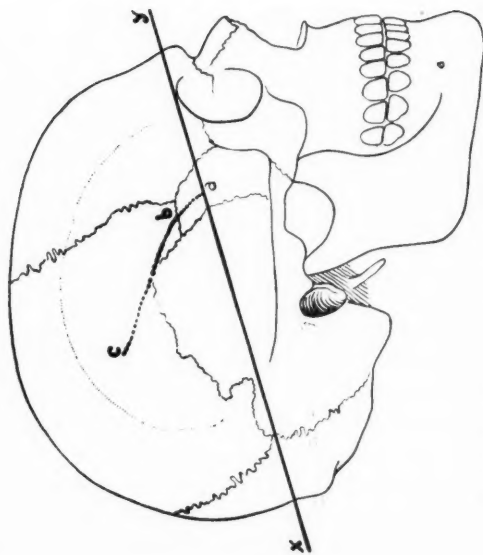


Fig. 3. Outer surface of cerebral hemisphere of human embryo of about five months. (After Gray.)



Dotted line shows posterior ramus of Sylvian fissure.

Fig. 4.

safe or desirable to turn up or down a dural flap for inspection of the cortical structures beneath, we would still be at sea; for even the great interlobal clefts or fissures as they appear on the brain cortex are not sufficiently differentiated to be easily identifiable except by their relation to the brain as a whole.

A very little study on the cadaver will show that the outer cortical cleft of the Sylvian fissure starts, and must start, on a level with the posterior margin of the floor of the anterior cranial fossa, which fortunately is not difficult to locate.

Guide.—Locate by palpation the superior margin of the orbit and the fairly distinct bony ridge marking the anterior boundary of the temporal fossa. Draw an imaginary line upon the skull the anterior end of which is tangent to, or passes through, the superior orbital margin, the posterior end leaving the skull at a point just above the external occipital protuberance. A point on this line, roughly $1\frac{1}{4}$ inches behind the anterior temporal ridge, marks the position of the posterior margin of the floor of the anterior cranial fossa and also the cortical starting point of the Sylvian cleft or fissure. (Fig. 4.) From point *a*, the Sylvian fissure curves sharply upward to point *b*, from which region the three rami diverge. At point *b*, the fissure turns slightly downward, still continuing, however, as the posterior ramus a course backward and upward. Through and at right angles to this direction line three incisions should be made.

As to the depth of incisions: theoretically it is clear that any inflammatory reaction following the knife in the submeningeal cerebral tissues might tend by pressure to react unfavorably upon the cortical subarachnoid outlets. Nevertheless, it should be considered. Theoretically, and probably actually, mere dural incisions are obviously insufficient. In the posterior fossa region—i. e., through the inner wall of the obliterated sinus—I would advocate shallow incisions but of sufficient depth to insure opening of the subarachnoid space. A depth of 5 mm. should insure this. Through the line of the posterior ramus of the Sylvian fissure it would seem to me that deeper incisions—i. e., to a depth of half of half an inch—might be employed as providing longer and perhaps more adequate drainage outlets. (Figs. and 6.)

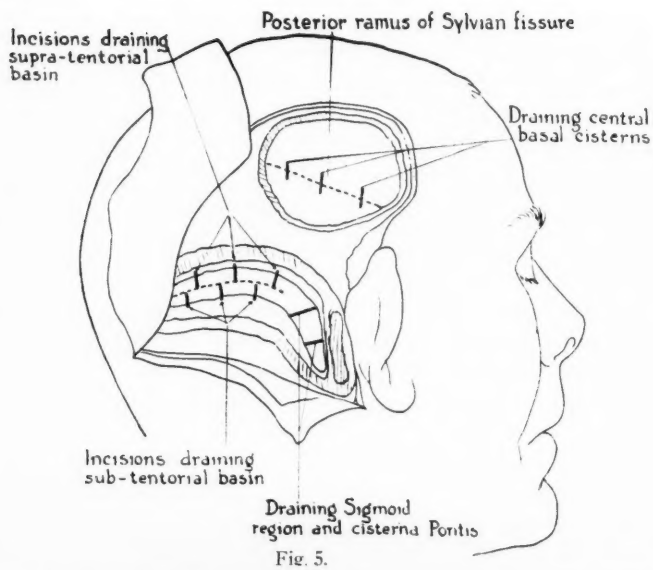


Fig. 5.

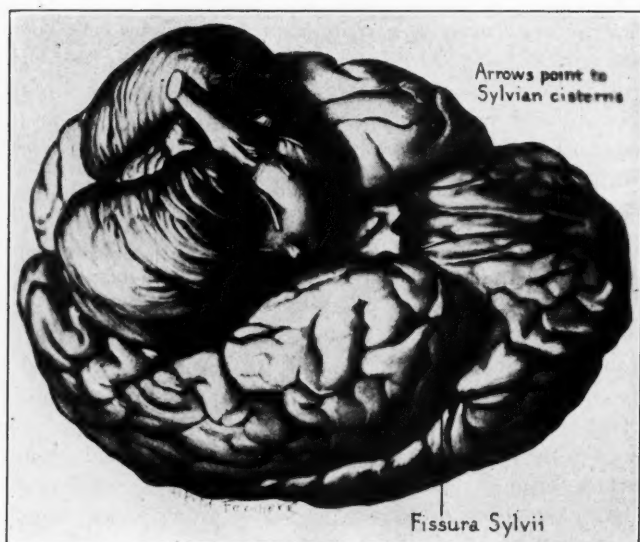


Fig. 6.

I wish to repeat here that I have suggested this topographical scheme chiefly as a basis for investigation and animal experimentation. Therapeutically it is admittedly only a theoretic hypothesis, but a hypothesis which would seem to offer fairer promise of therapeutic results than any surgical effort having no logical relation to the gross pathology of the disease. The so-called forced drainage theory of Kubie, the physiologic reactions to which have been definitely established, is also as yet only a theoretic hypothesis, but with a very logical basis and exceedingly promising implications. While, therefore, we are considering these two hypotheses, let us allow our imagination to play with their combined possibilities. Suppose (1) that animal experimentation should prove that this or some better scheme of direct surgical drainage is not of itself necessarily dangerous to life; suppose (2) that animals which had been subjected to subarachnoid inoculation with pathogenic micro-organisms and subsequently operated upon by the method here suggested, exhibited some recoveries as compared with the fatalities of inoculated control animals not so treated; or again, suppose that no results were obtained; and finally suppose (3) that these experiments were repeated, the surgical provision of drainage being supplemented by the Weed-Kubie method of intravenous injection of a properly proportioned hypotonic solution; that this combined method resulted in a relatively copious and sustained discharge of cerebral fluid through the surgical outlets and an appreciably increased percentage of recoveries. We would then have made a definite therapeutic advance based on a demonstrated reduction of mortality.

The history of surgery is punctuated by outstanding triumphs over the fatalities of disease. Seventy years ago hundreds died of appendicitis who would be saved through surgery today. Less than fifty years ago, scores died of septicemia resulting from infective sinus thrombosis. Thirty-odd years ago many died of the indirect results of suppurative labyrinthitis without even a tentative diagnosis of the initial lesion having been made. It required clear and courageous thought to conceive and execute the first successful operation of appendectomy; of combined clot removal and jugular ligation or resection; of labyrinth exenteration. It may still require years of patient investigation to bring

otitic leptomeningitis within the category of lesions relatively under surgical control; but I predict that this also will come. It will come the sooner and more certainly if we study and restudy the work of Flexner, Weed, Wegeforth, Kubie, Cushing, Fremont-Smith, Putnam, Cobb and others, and discard any theories or measures which run counter to sound physiologic law and established scientific fact.

Supplementary Note.—In informal discussions it has been called to my attention that, though this paper suggests only animal investigations of the two theses proposed, it is not impossible or unlikely that in the difficult problem presented by otitic meningitis the surgical measure here outlined may be tried upon human subjects. I can conceive that in consultations of surgeons over a given case this might be decided upon. It is my opinion that the surgical scheme proposed should not be tried except in conjunction with the so-called forced drainage method of Kubie. The latter measure, preferably by intravenous injection of a properly proportioned hypotonic solution (never of distilled water) should be instituted promptly after completion of the surgical operation. The importance of this in stimulating cerebrospinal production and flow from the perineural spaces of the central nervous system, in maintaining the patency of surgical outlets, and in preventing obstruction by uneven distribution of pressure through collapse at certain points, is so obvious as hardly to require argument. As with every surgical theory of treatment in otitic meningitis, the earlier it is decided upon and executed, the better should be the chances of a successful outcome. Should any surgeon take it upon himself to try this method, he will confer a favor upon me if he will send me a brief line reporting the result, whether favorable or the reverse.

10 EAST FORTY-THIRD STREET.

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DISCUSSION—DR. LAWRENCE S. KUBIE, NEW YORK.

1. Dr. Kerrison has spoken to you of "forced drainage." It must be evident to all that it is impossible to talk of forced drainage unless one has a continuity between all of the different fossæ, ventricles and cisterns of the intracranial spaces. As is well known in severe inflammatory conditions of the central nervous system and its coverings, it frequently happens that the various compartments of the intracranial spaces become walled off one from another. When this has happened it is impossible to establish any really adequate drainage by a single point of outflow for the fluid. It is therefore essential to attempt to establish drainage as early as possible in the course of the infection.

2. It must also be emphasized, however, that it is essential, not merely to drain the surface, but the depth of the central nervous system as well. In this connection it must not be forgotten that even an early and superficial meningitis is accompanied by very much more extensive involvement of the mesodermal sponge-work, which penetrates through every nook and cranny of the

central nervous system, than is ordinarily taken into consideration. There is an accumulation of inflammatory products deep in the central nervous system from the start.

3. It is natural enough to think first of a wide opening of the cranium or of the spinal column as a method of securing drainage. Unfortunately, however, this does not take account of the fact that such a wide opening of the bony coverings of the central nervous system allows a collapse of the meninges to occur; and even more serious than that, it sometimes allows partial movement of the whole mass of the brain, a settling on the base, or a movement through the falx cerebri to one side or to the other, or backwards through the opening in the tentorium. As a result of such displacements of brain mass, there can be partial obstructions which will interfere with the outflow of fluid and prevent the very drainage for which the opening has been made. Then one is faced with a situation like that in which an abscess tract has collapsed, leaving loculated pockets of pus behind.

4. It is therefore essential, not merely to drain the infected cavities, but to prevent the collapse of the meninges and the displacement of brain mass by constantly and continuously forcing the new production of cerebrospinal fluid. It is this which constitutes the essence of the method of forced drainage.

5. It has been demonstrated fairly thoroughly by experimental work that when fluid production is accelerated by a lowering of the osmotic pressure of the blood, at the same time that free escape of the cerebrospinal fluid is allowed, such new-formed fluid is made, not only at the choroid plexus but by transudation through all of the capillaries of the central nervous system. Therefore such fluid does not merely wash the ventricular and subarachnoid systems, but works its way out along the perivascular channels from the depths of the central nervous system to the surface. There is good physical and physiologic grounds by which to understand this. Within the cranium, under ordinary circumstances, fluid is transuded through capillary walls in the face of a high intracranial pressure and is again reabsorbed at the venous end of capillaries under the influence of that pressure. This is in keeping with the old and fundamental work of Starling on fluid

transudation through capillaries. As a result of this positive intracranial pressure there is never an excess of fluid in the pericapillary spaces over that which can be reabsorbed at the venous end of these capillaries. If, however, the intracranial pressure is reduced to atmospheric pressure by drainage, and if at the same time the rate of formation of cerebrospinal fluid is stimulated by lowering of the osmotic pressure of the blood, there is forced a great increase in fluid production over that which can be absorbed, and as a result this fluid must find its way to the surface along these perivascular channels.

In this way it is possible to get fluid movements from the depths of the central nervous system to the surface and out through whatever drainage tract is provided by the operator. Such fluid movement has been shown experimentally to cause a washing out of the products of the inflammatory reaction from the depths of the central nervous system and out the drainage tract entirely. It is this free extrusion of lymphocytes from the depths of the perivascular spaces under the influence of forced drainage which produces the phenomena to which Dr. Kerrison has referred in his paper, to wit, the change in the cytologic picture in the fluid from a predominant polynucleosis to a predominant lymphocytosis. These lymphocytes come from the depths; the polymorphonuclear leucocytes chiefly (though not exclusively) from the surface.

6. There are various methods of drainage which have been tried. No one of them is perfect and all of them present difficulties. This problem has to be worked out individually with each case. Spurling, in Louisville, Ky., has done a laminectomy on a single lumbar vertebra under local anesthesia, inserting a cigarette drain, and treating the whole as a surgical wound. By weighing the dressings, he has been able to ascertain that with forced intake of fluid by mouth and intravenously, he could increase the fluid output over a period of one or two weeks up almost to one or two liters a day. Retan, in Syracuse, has used an ordinary lumbar puncture needle on children, and kept the child for many hours on a Bradford frame. At all events, it is clear that the first point of drainage, other things being equal,

should be the lumbar region with the head high so that one drains the entire tract as fully as possible.

It must be emphasized that such drainage alone, in the presence of an active focus of infection, cannot possibly result in a cure. Furthermore, whenever adhesions have already separated the intracranial cavities into independent compartments, drainage through the lumbar route will be insufficient. In either of these cases forced drainage must be supplemented by surgical procedures such as those which are described by Dr. Kerrison.

7. The best method of promoting fluid formation is undoubtedly the method of intravenous injection of hypotonic solution. These solutions must contain just enough salt to prevent hemolysis of the red blood corpuscles. If such injections are made very slowly no increase will occur either in venous pressure or in intracranial pressure. The fact that no increase occurs in venous pressure is of great importance, because it shows that the fluid is put out of the blood stream into the tissues as rapidly as it is injected, when this injection is made slowly, and that no overloading of the circulation occurs and no strain upon the heart. The fact that no material increase in intracranial pressure occurs is of obvious importance when one is dealing with infections of the central nervous system. By means of such injections, even when very conservatively applied, it has been possible to increase the fluid production more than ten times. Whether such fluid is called a filtrate or a dialysate is obviously, therefore, from the point of view of practical application, a secondary matter. Yet it may be of some importance, however, in a subsidiary way, as indicating the possibility of carrying over drugs and immune bodies from the blood stream to the cerebrospinal fluid. This, however, is a matter which cannot be discussed here.

8. There are certain obvious limitations and difficulties in the way of the application of this method of treatment. In the first place, of course, there must actually be free drainage. Without such drainage the method has no chance of exerting its action. There are two main obstacles to free drainage: first, the excessively thick and gelatinous exudate which forms rapidly in the course of certain types of infection of the meninges (as, for instance, in certain forms of pneumococcus infection); and sec-

ondly, the formation of encapsulated foci and pockets of infection which continuously feed infection into the rest of the system without ever becoming drained themselves. With regard to the first (the thick exudate), it is not possible as yet to say to what extent it is possible to so dilute this exudate by forced drainage as to render it more mobile and free to drain. This will have to be tested out clinically. With regard to the second, there has recently been reported a case which points to a possible method of treatment. A few weeks ago Dr. Leon Cornwall reported a case of obstinate meningococcus meningitis in which the fluid could repeatedly be made to come clear as a result of a combination of drainage and intraspinal use of serum, but which nevertheless repeatedly relapsed. Finally he injected air into the subarachnoid system, and apparently by so doing broke up adhesions to such an extent that there was a discharge of a great deal of pus which had not previously been evacuated and after this a prompt recovery of the patient. This case was reported at the May meeting of the New York Neurological Society. It would obviously be possible in resistant cases to interrupt the periods of forced drainage by an injection of air with an effort to break up adhesions and thus increase the free passage of fluid from one portion of the subarachnoid and ventricular spaces to another.

9. Promising results have been reported in a variety of conditions, to wit, from Boston there come reports of extraordinary results in optic neuritis, from Louisville (Spurling) and from Syracuse (Retan) there have been reports of favorable results in poliomyelitis, early infantile syphilis of the central nervous system, and in a variety of different forms of purulent meningitis, and perhaps in tuberculous meningitis. It is not possible, of course, to draw any conclusions beyond the very conservative and tentative one that so far the results are promising. No one kind of illness, and no one type of infection has been observed often enough to put it on the statistical basis without which a final evaluation of the utility of the method cannot be made. It is Dr. Kerrison's hope, as it is mine, that, as a result of this paper, enough people will become interested in the problem and in the method to give it a trial while at the same time subjecting it to rigid control and criticism.

10. And finally, I would like to include one comment which does not bear directly on the problem of forced drainage, but rather on the whole question of intracranial surgical drainage. It seems to me that it would be worth while to consider using the nasal cavity as a point of approach to the base of the brain. It is well known that in fractures through this region it is possible to get very extensive leakage of cerebrospinal fluid with spontaneous replacement of fluid by air. Cushing has shown that the same thing can occur in orbito-ethmoidal osteomata. Here is a pathway by which continuous drainage could be very readily established during the course of treatment by forced drainage.

LIV.

IS ALL HEARING COCHLEAR?*

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Since the time of our college days most of us have probably been accustomed to consider the VIIIth cranial nerve as consisting of two divisions, a "cochlear" and a "vestibular," the cochlear division being concerned with hearing and the vestibular with bodily posture and equilibrium. Of the two names the first, which denotes that the nerve fibers in question come from the single receptor organ of the cochlea, is the more precise and distinctive. The vaguer, less determinate term vestibular is left to do compound duty and to cover the whole remaining set of nerve fibers that connect with the saccular macula, with the utricular macula and with the three cristæ of the semicircular canals.

While it is eminently convenient and necessary to have a terminology that discriminates between the auditory and the equilibrium functions of the VIIIth nerve, we must at the same time be reasonably certain that our line of division is drawn at the proper place. In this connection two comments of a general nature may be made at the outset. Had the physiologists who introduced these names been dealing with the nerve supply, not of the mammalian labyrinth but of that of lower vertebrates, they would have had no ground for using the word cochlear at all. The cochlea is purely a mammalian organ. Meantime fishes, amphibians and reptiles have neither cochlea nor organ of Corti. Instead, they have in the region occupied by the cochlea of mammals at least one additional otolithic receptor, the lagena, not to speak of still others that we need not here discuss. The second comment is this. While it has been fully established that the semicircular canals and the utricular macula of all vertebrates subserve an equilibrium

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function, the case is by no means so clear with respect to the saccular macula. To my knowledge, there is only one experimentally based claim that this receptor influences posture—Benjamins and Huizinga (1927) found that its ablation interferes with certain eye movements in the pigeon. On the other hand, the following investigators failed to establish any connection between saccular macula and posture: Parker (1909) and Maxwell (1923), in the case of the dog-fish; Laudenbach (1899), McNally and Tait (1925) and Huddleston (1928) in the case of the frog; and Versteegh (1927) in the case of the rabbit. To this list may be added an unpublished finding by Dr. McNally and myself on reptiles: ablation of the succules in a rattlesnake causes no detectable disturbance of equilibrium or of posture.

Throughout the past three or four years, Dr. McNally and I have been engaged on a detailed reinvestigation of certain parts, particularly the equilibrated region, of the frog's labyrinth. In numerous respects we have succeeded in obtaining fresh light on the action of each receptor (and of each type of receptor) in the intimately associated group. During the course of this work the nerves to the saccular maculae have again and again been cut. In no case could we establish any relation of the saccules to bodily equilibrium or posture. This persistent failure to ascribe any postural function to the saccular receptors naturally gave one to think, and this is how the matter presented itself.

We know of two, and as yet only two, functions of the inner ear, namely, audition and equilibration. In its auditory aspect, the eighth nerve subserves an exteroceptive function—i. e., it responds to some happening which, arising in the external environment, impinges upon the animal from the outside. In its equilibrated aspect the same nerve appertains to the proprioceptive category—i. e., it is concerned in signaling internal change due to the animal's own movement. Can one form any reasonable conjecture as to the process by which, in the course of evolution, proprioceptive function and hearing become so closely linked together?

If one assumes that the eighth nerve in its primitive beginning was auditory, there would seem to be no rational or imaginable way of deriving therefrom or of superadding thereto an addi-

tional proprioceptive activity. If, on the other hand, one sets out with the supposition that originally the nerve terminals subserved a proprioceptive use, it is easy to imagine a process of change or of differentiation whereby they might come to acquire auditory attributes.

The typical receptor of the labyrinth, if one may use such a phrase, is otolithic. Now, it happens that otolithic receptors are very widely distributed throughout the animal kingdom. Whereas canals belong exclusively to the vertebrate phylum, and whereas an organ of Corti is quite a late vertebrate acquisition, a large number of invertebrates possess otoliths. Indeed, if one makes a survey of the distribution of otoliths among animals at large, one is driven to the conclusion that this type of receptor, like eyes, phosphorescent organs, etc., has been invented over and over again and by wholly different animal stocks. The otolithic, in other words, is a fundamental and extremely common type of sense organ.

We all know of Kreidl's experiments on the otocysts of the prawn, whereby he showed that the otoliths—in this particular case grains of externally collected sand—are concerned in imposing direction with respect to the field of gravity. Another crustacean example, equally worthy of mention, is that of the delicate little swimmer known as *Mysis* (Fig. 1). This creature, which belongs to a different group of crustacea from the animal used by Kreidl, carries a pair of elaborately constructed otocysts in the telson or terminal segment of its tail (Fig. 2). Removal of these sense organs brings about two disabilities. The first is that the animal becomes disoriented and no longer swims back up. The second disability is of especial interest to us. *Mysis* swims in shoals in the sea water near the shore. Suppose one nets a number of the tiny creatures and places them in a wide bottle or glass aquarium. Each time that the side of the container is tapped with the finger every animal of the shoal makes a sudden side-ward or backward leap in the water. The reaction is no doubt a protective flight reflex that serves to drive the animals from regions of wave-break or other dangerous water disturbance. A *Mysis* deprived of otocysts shows no such reaction. In this case, therefore, one and the same otolithic organ serves a double func-

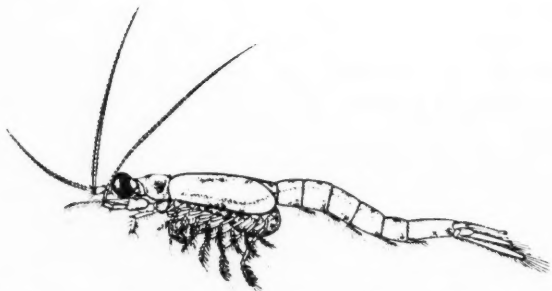


Fig. 1. *Mysis relicta*, enlarged about three times. (From Calman after Sars.)

The otocysts are in the tip of the tail.

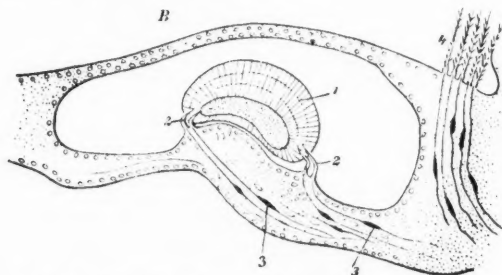


Fig. 2. Magnified section of an otolith organ in the telson of *Leptomysis gracilis*. (From Mangold after Hesse.)

1, the otolith; 2, sensory hairs; 3, sensory nerve cells.

tion. In its orienting capacity it acts as a proprioceptor; in its response to succussion or vibration it is an exteroceptor, and at that an exteroceptor essentially of the auditory kind. It signals water tremor or water vibration.

Much laborious discussion has centered around the mode of action of our cochlear apparatus. By way of pleasant variation, let us turn for a moment to consider the simpler case of an otolithic organ.

First with regard to its gravity-directional action. As the name would suggest, every "ear-stone" or otolith has a higher density or greater specific gravity than that of the surrounding fluid. It thus at all times exerts residual weight in the downward direction. The appended diagram (Fig. 3) will show how a mere loose

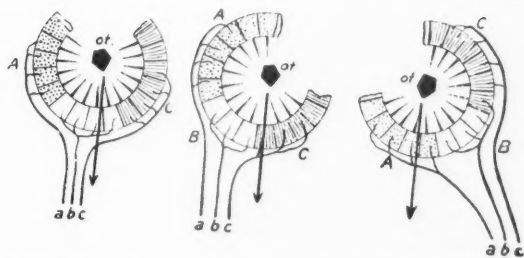


Fig. 3. Diagram of an otolith organ, to show how alterations of its position will cause the weight of the otolith (*ot.*) to press on different sensory cells, and so to affect different nerve fibres. (From Starling.)

A, B, C, different diagrammatic sections of the otocyst cavity; *a, b, c*, the nerve fibres that correspond respectively to sections A, B, C.

sand-grain, as in the case of the prawn, might suffice to compel a proper orientation of the animal with respect to gravity. As a rule, however, otoliths are made by the animal itself and grow with its growth. In these circumstances the otolith, like the lens of the eye, is an integral and duly incorporated part of the total, more compact receptor organ. In the case of *Mysis*, for example, and in the case of the utricular receptor of vertebrates, it fits upon and corresponds in size with its underlying patch of sensory epithelium. All the indications point to the fact that these gravity-directing otolithic organs do not signal continuously, but only when, by unusual inclination of the animal's body, the otolith is allowed to slip—forward, backward or sideward, as the case may be. Some relative displacement between the otolith and the sensory cells—for convenience we shall call this displacement a slip—is necessary in each case to excite the nerve terminals and thus to bring about an appropriate readjustment of the body.

It is perhaps easy to see how one and the same or another similar otolithic organ (one and the same in the case of *Mysis*, similar in the case of the saccular macula of a fish or of a frog) might likewise respond to vibration set up in the water. The propagated tremor shakes the water-encompassed animal. Meantime the otolith by inertia tends to stay still. The result on this occasion is a continuous or periodic stimulation of sensory cells analogous to the cochlear events that accompany hearing.

Hitherto my object has been to indicate that an otolithic receptor, originally designed, let us say, in the interests of simple bodily orientation, might, at least in the case of aquatic animals, take on an auditory function as well. The use of the word auditory in this connection leads in turn to a brief consideration of what the physicists include under the term sound.

When we commence the study of physics we make for ourselves certain enlightening, albeit rather unsettling, discoveries. To take one example, radiant light and radiant heat are revealed to be essentially the same kind of thing, notwithstanding the fact that the bodily apparatus for their respective detection, and also the corresponding sensations in each case involved, are widely different. That department of physics known as sound contains its own surprises. The pity is that the physicists themselves have as yet hit on no better word than "sound" to cover the broad range of phenomena concerned. By Sound, whether written with a capital letter or not, the widely generalizing physicist means, not what you and I commonly understand by the term, but the propagation or transmission of all molecular elastic vibrations, through gases (including air), through liquids or through solids. Using a common mathematical method to deal with them all, and blandly ignoring the variety of sensory mechanism employed for their detection under different circumstances, he asserts that the mere medium of transmission makes no real difference to the essential phenomenon. At whatever disrespect to our cochlea or to our more sacred feelings as otologists, let us for once—but not for long—affect this physical mode of speech and reckon sound as a special type of external event that takes origin and runs its course in solid bodies, in liquids, or in air.

Mundane Nature is simply full of sound. Few events happen on the surface of our globe that do not simultaneously set up vibrations in some body or other, whether in rock or in timber, in water or in air. Every raindrop that falls from the sky, every stream that hurries down the hillside, every wave that gathers and breaks, every branch or twig that falls from a tree, every obstructed gust of wind, every animal that steps on the solid ground or lands after a leap, sets up in the surrounding material

environment, whether this be solid, liquid or gaseous, a whole series of molecular oscillations, violent or insignificant, as the case may be. From their original focus these are promptly conducted in different directions, to die down in the end like the ripples from a splash in a still lake. All around us perpetual shakes and quivers are occurring, and it is a matter of accident, as it were, that by far our best receptor for their detection is the cochlea, which is sensitive to vibrations only of the air.

You will probably concede that most of the more abrupt tremor-accompanied happenings, such as storm, water-splash, cracking of branches, tree-fall—sudden impact of whatsoever kind—are apt to be of vital concern to animals at large. All members of the cat tribe, for example, have pads on their feet for the express purpose of minimizing vibration. It is, therefore, a matter of physiologic concern that animals should become possessed of receptors for picking up these transmitted disturbances. That trout, for example, have a delicate sensory equipment for reception of under-water vibrations that may fail entirely to affect our ear, is known to every fisherman. Naturally an aquatic animal or, let us say, even a spider, may have no apparatus that responds to the purely air-borne vibrations which give us the characteristic sensation of cochlear sound.

When we come in this fashion to deal with natural history at large, an additional and very important fact emerges. Not only are animals, in the interests of individual self-protection, sensitive to externally arising tremor, but many of them, for purposes of racial preservation, have contrived to utilize and to take advantage of that marvelous physical property of solid and of liquid bodies (not to speak of air) in virtue of which tremors are so readily conveyed from their place of origin to a distance. For sexual purposes—that is to say, for attraction of distant partners, they gradually devised means of themselves giving origin to propagated tremor. What began in this humble way has ended in the greatest social contrivance known in mundane affairs, namely, our own faculty of articulate speech.

Please pardon me if I begin with the frog. At this season of the year Aristophanes' *βοεξεξεξέξ κοάξ κοάξ* resounds in every pool of the countryside. We in America are perhaps especially

avored in the matter of repertoire, because the ranine chorus ranges all the way from the deep bass of the bullfrog to the clear, birdlike pipe of the little tree-frog. Each male representative of whatsoever species at this time of the year comes nigh to bursting in the pure physical effort of projecting to the utmost distance his characteristic vocal signal. I use the somewhat neutral word "signal" advisedly, because it is doubtful whether the amatory invitation ever touches the coy female by transmission through the air.

Professor Yerkes (1905) was at elaborate pains to discover whether the frog has a sense of hearing. He found, for example, that frogs perched on the edge of a ditch or on the bank of a stream are not startled by the loudest noises, though on the slightest visual alarm they promptly plunge into the water. By a complex arrangement of laboratory apparatus he did eventually to his own satisfaction make evident that noise is not wholly a matter of unconcern to the animal. On critical examination, his ingenious experiments lack just one final touch of rigor. He used an electric bell as a source of sound. He fails to state, however, what precautions were taken to prevent the rattle of the bell from being conveyed through the apparatus itself to the animal under observation. An electric bell not only gives out air-borne sound, its buzz communicated along a table-top may even be felt at a distance with the fingers.

Entomologists tell us that the ominous ticking of the death-watch is merely a love call. The rapping is done by the male animal and serves to attract the female. That we should hear it, no more proves that the female insect has a physiologic equivalent to our cochlea than does the audible shrilling of the male cicada prove that the female of the same species receives the sound through the air. For all we know to the contrary, most of the sound signals emitted by insects may be directly applied to wood (or woody stem), handed on by wood and by wood delivered to the recipient.

Let us, however, come back to the frog, with which we ourselves as members of the vertebrate group have closer affinity. For some reason as yet unexplained the operation of double labyrinthectomy makes the animals particularly prone to croak.

The croak in this case is not spontaneous or continuous, as in the case of pond frogs at the breeding season. It is induced only by stimulation, but the type of stimulation is so simple that it is all but impossible to handle the animals without eliciting a croak. Repeated observation has convinced us that the condition necessary in these cases to evoke the response is some externally conditioned displacement of the spinal vertebrae with respect to one another. As any accidental impact or any handling, no matter how careful, is apt to bend the spinal column, it is difficult to maneuver the animals in any way and at the same time keep them silent. If one is picked up and held between the fingers it croaks unceasingly, but in this case we have been able to show that the felt movement of the abdominal muscles causes on each occasion a corresponding, wholly irrepressible and wholly involuntary clutching or converging action of the experimenter's fingers. It is this unintentional thumb-and-finger jerk that keeps the music going. A frog similarly suspended between the jaws of a metal clamp does not continue to croak. As the specimens are at all times well filled with air, they float, either prone or supine, on the surface of water. In the depth of a Canadian winter it is a curiously pleasant thing to have in the laboratory three or four of these frogs floating in a tank with the water-tap gently running. As the animals frequently move and bump each other or then sail slowly under the tap, where they are similarly thumped or pounded by the falling water, they send out at frequent intervals their cheery music, giving one almost to believe that spring has arrived.

The frog is peculiar in having a long backward extension of the membranous labyrinths on each side of the vertebral column. Associated with these extensions is a chain of otoliths, the well known "calcareous bodies" of the Anura. Whether these otoliths, as is quite likely, are the real receptors for reflex production of the croak, we cannot as yet say. Versluys (1929), professor of morphologic zoology in Vienna, has recently made a survey of the distribution of calcareous bodies within the group of frogs and toads. He believes that those members of Anura which are devoid of calcareous bodies are poor jumpers, and in consequence he suggests that the organs in question are concerned with locomotion.

tor function. As the otolith-bearing extensions of the labyrinth appertain to its posterior rather than to its anterior (equilibrical) part, it is just as likely that they may be found eventually to coincide with the possession of croaking ability.

In discussing the apparatus for possible reflex elicitation of the croak, I have been dealing with a side issue rather than with the question that more directly concerns us. Purely by accident we found, while handling delabyrinthized frogs, that the croak sets up a powerful under-water disturbance. If one dips one's finger points into the water in the immediate vicinity of a croaking animal, one experiences a sensation not unlike that which results from touching the terminals of an electrically active induction coil provided with a vibratory interruptor. The water tremor thus set up has such a smiting, stinging effect that it is capable of registering an influence even on the skin receptors of one's fingers. If one uses a hydrophone (a binaural stethoscope whose open end is covered with rubber dam) to listen to the under-water tremor, one finds that at the distance of a centimetre from the animal the noise is unbearable. It causes actual pain of the ear drums. As the hydrophone is moved successively farther and farther away the intensity of the noise dies down, at first with rapid gradient and then by less sharp steps. At the greatest distance that we have been able to obtain in the laboratory (about three metres) it remains perfectly audible.

Plainly the croak that we pick up by way of the air is also carried by another medium. Let me here remark that in producing his vocal signal the frog emits no air from the anterior respiratory passages as we do in speaking. The air contained within the lungs is merely shifted temporarily to the mouth cavity and to the capacious air sacs beside the mouth; in the intervals between each act of croaking it is silently passed back to the lungs again. The proof of this is that a frog, pushed entirely underneath the water, will continue on appropriate stimulation to croak as well and as long as before; meantime no slightest bubble of air escapes from the nostrils. As heard by the hydrophone the croak of the submerged is just as powerful as that of the surface-resting animal. Yet to the unaided ear listening above water the noise is all but imperceptible. That the frog should ordinarily

emit his croak with nose and back above water is due merely to the fact that when physiologically adjusted for croaking he is hydrostatically lighter than the water. His simple buoyancy keeps him partially in the air medium, and so we are able to hear him.

During the war, for purposes of intercommunication between submarine vessels, the need arose of having a transmitter sufficiently powerful to propel its messages through long stretches of water. Whereas adequate receiving devices (hydrophones) were soon developed, the problem of designing an apparatus that can produce long-distance subaqueous tremor is more difficult, and at least one government is reputed to have spent considerable sums in unsuccessful experiments to this end. Finally Dr. Louis V. King of McGill University, having carried out the necessary preliminary calculations, built an instrument which on the very first trial came up to specification. It would transmit to a distance of twenty miles. His apparatus consists of a heavy metal plate that can be electrically set in vibration. I do not know whether the principle of the frog's apparatus for under-water signaling could be adapted to meet the heavy demands of war-craft, but the question is at least worth considering. The frog has, as it were, two compartments for holding air. These are separated by the narrow and rigidly held chink of the glottis. While the latter acts as a sounding reed, the anterior compartment, consisting of mouth and air sacs, serves the same function as the expanded plate of a loud speaker. By its mere extent of surface it serves powerfully to reinforce the emitted sound. This can be proved by listening with a hydrophone to a simple pipe reed that is sounded under water. The noise so produced may be feeble in the extreme. So soon as one affixes over the end of the pipe a thin rubber bag which becomes inflated as the pulsating air rushes in, the apparatus is suddenly raised to a new and astoundingly effective level of output. Meantime its energy requirements remain just as before. We thus come to recognize the true function of the laterally situated mouth sacs of the croaking frog.

While delabyrinthized frogs croak only in response to gross mechanical stimulation applied to the region of the back, there is no doubt that intact males at the breeding season also croak

in response to the water-transmitted croaking of other males. I once had an opportunity in Italy of observing the habits of the green tree-frog (*Hyla arborea*) in the breeding season. At this period these little animals take up their quarters in pools of water. A short, shallow ditch may contain a dozen or more. Their voice—for one can scarcely speak of a croak in the case of these diminutive choristers—is a vibrant high pitched note of charming musical quality, which, continuously and rapidly repeated, at first deceived one into the belief that some bird was calling in the neighborhood. When one approaches the ditch whence these clear toned notes proceed, the music suddenly ceases and stillness reigns. Not a movement or ripple on the water betrays the location of a single frog. In perfect concealment among the green weeds and grasses, the isolated males sit without even troubling to submerge. It was only after some experience that one learned how to detect and capture them, but the method adopted proved to be infallible. Leaning over the ditch and looking into the water one remains motionless. After a minute or two a distant frog, apparently reassured, starts to pipe. In another moment another nearer frog commences. Then suddenly, right under one's nose, it may be, an animal hitherto invisible becomes piercingly vocal. By this time the whole male chorus is once more in full action. The impulse under these conditions to pipe, even in the presence of possible danger, is simply irresistible. As the little creature sits at a distance of a few inches, the water surface below its chin is thrown into tiny ripples by the vibration of the pharynx. Glancing along the level surface of the water, one sees similar ripple centers at various points among the grasses. The lurking place of every member of the chorus is thus revealed, for one's unaided auditory sense is remarkably defective in localizing the source of their piping voice.

At the time one supposed that the sequence in taking up the piping was an affair of rivalry and that each frog, unwilling to be outdone, was striving to outdo its neighbors in vocal effort. The fact that a distant frog first took up the chorus was explicable on the ground that the nearer animals had originally been most disturbed by one's approach. But the supposition of relative degree of original disturbance entirely failed to account for the

almost instantaneous reply and utter lack of hesitation of the nearest animal to take up the melody, once its turn arrives. Plainly each successive frog, after a certain brief latent period, is tremor-stimulated by its neighbor. They pipe, not necessarily in rivalry nor in response to simple auditory stimulation, but because, with a receptive mechanism that is very responsive within limited distances to water-conducted tremor, they are forced so to do. The fact, too, that all the congregational croaking of frogs takes place in water, and the further fact that the chorus, once started, rapidly swells in volume, to subside occasionally and to rise by successive accumulation again, means that only in water do the conditions exist for adequate tremor stimulation of one animal by the other.

The real purpose of the croaking apparatus, however, is to attract from a distance the ripe females. One observer, Courtis (1907), finding some copulating toads in a shallow bay in Lake Michigan, separated the females and removed them to a distance. He noted that members of either sex could in the water locate the direction from which the vibrant, trilling note of an isolated male toad proceeded and swim in that direction. The directed approach of the females occurred even at a distance of 30 or 40 feet. It is to be noted that, unlike *Hyla arborea*, the individual male toads also responded to each other's call. "When there were twelve unattended males within four or five feet, a call by one of their number would bring the others from all directions, and in a second or two there would be one or two heaps of clasping, fighting, kicking males, squealing like mice, and rolling over and over." While the written account shows that the observer was thinking in terms of air-conveyed rather than of water-transmitted guidance, he was nevertheless able to prove that it is the sound that gives direction to the toads. While the animals were engaged in forward swimming, at which time they do not croak, there was no attraction, and separated males and females might swim past each other without any apparent recognition.

Having dealt with the frog, we shall now for a space turn to fishes. It is a common observation that some fishes on being removed from the water emit audible sounds. In most cases, as Dufossé (1874) first showed, the sound is produced by the air

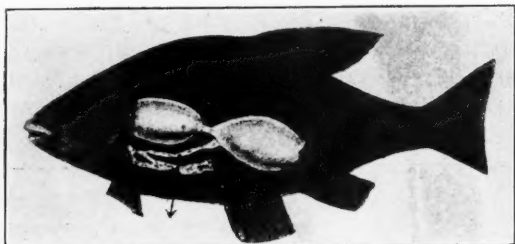


Fig. 4. The air-bladder of the tench. (From Thilo.)

Note the two compartments separated by a constriction. An arrow is drawn downwards from the duct that connects the posterior compartment with the pharynx.

bladder. As you may know, there has been considerable discussion as to the function of the air bladder in fishes. Sometimes, and especially when it is a closed sac, it acts as a hydrostatic organ. There is, however, one outstanding group in which the air bladder is decisively a tremor-emitting apparatus. This is the subclass of teleosts known as the Cypriniformes or Ostariophysi, the members of which mostly live in rivers or in lakes, and frequently inhabit rather muddy water. In these forms the air bladder is peculiar in a number of respects. To begin with, it consists of two compartments, separated by a constriction (Fig. 4). The posterior compartment communicates with the pharynx by means of a duct. A chain of ossicles, the so-called Weberian ossicles, (Fig. 5), connects the front of the anterior compartment with the labyrinth. By means of muscles, whose distribution and mode of attachment differ in different representatives of the group, the air within the anterior compartment can either be thrown directly into vibration or then made to vibrate by being forced through the constriction that separates the compartments.

Whatever the method of producing the sound, there is every reason to believe, as Sørensen (1895) especially claimed, that all members of this group communicate with each other by means of the air-bladder apparatus. This author, who studied South American forms, reports that during the breeding season the babel of noises made by different species is loudly heard by an observer stationed in the hold of a river vessel. If he steps on deck and

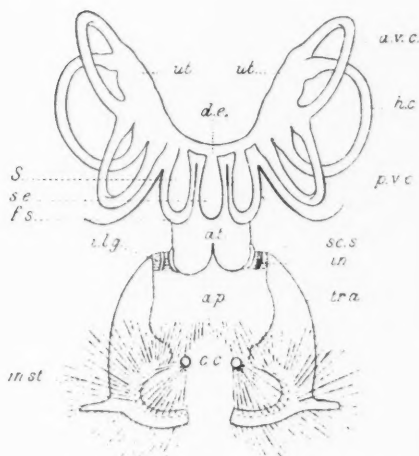


Fig. 5. Diagrammatic representation of the Weberian ossicles showing their relation to the labyrinth in a siluroid fish. (From Bridge and Haddon.)

tr.a., anterior process of the malleus; *in.*, the incus; *sc.s.*, the scapes. The two membranous labyrinths of the cypriniform fishes are connected with each other across the middle line by a duct (*d.e.*), from which a medially placed sinus (*s.e.*) extends backwards. The vibrations of the two stapes ossicles are first communicated to the fluid in the perilymph chamber (*at.*), and thence to the medial sinus (*s.e.*).

listens from above, he can scarcely hear the multitudinous sounds that are perfectly transmitted to his ear through the sides of the hollow ship. No doubt the sounds thus produced serve to bring the sexes of different species together. Sørensen was also quick to point out that the selfsame air bladder that emits a certain kind of vibration is itself best adapted to receive and to resonate to that particular note when externally produced; in other words, that in virtue of a similar sound-producing and sound-receiving mechanism, fishes of one and the same species are doubtlessly attuned to each other. When we come to know more about the matter, it may well turn out, too, that different species of frogs within the same pool are similarly attuned each to its own peculiar type of croak. Any given species may be quite unresponsive to the croak of a different species.

E. H. Weber, who first carefully described (though he did not discover) the chain of ossicles to which his name is now attached, believed that they subserve an auditory function. Indeed, by analogy with the three auditory ossicles of mammals, he called the three principal members malleus, incus and stapes. While the bones are in no sense homologous with the members of the mammalian chain, the names he originally used have not yet been wholly suppressed. One can scarcely feel sorry for this. The fact is that those anatomists who have suggested new names for them—and they have been renamed more than once—have usually labored under the mistaken idea that they are concerned in signaling to the labyrinth, not sound vibrations, but the slow expansions or compressions of the air bladder that are presumed to accompany change of hydrostatic level (cf. Fig. 6).

The whole complex of apparatus which in these cypriniform fishes has been designed in the interests of sexual signaling may conceivably in some cases be used for family purposes as well. The common catfish (*Amiurus*) of our North American rivers is a member of the cypriniform group. On one occasion—this was before I had become specially interested in the subject of hearing—while wandering by the side of a Canadian stream, I was suddenly called to see a remarkable phenomenon. A dense black cluster of young fishes, in size and shape like unto a football, was slowly making its way through the somewhat turbid water. As it happened, I had a handled collector's net with me, and was able to fish up a large part of the living ball. This consisted of well over a hundred plump young catfishes. On reading the matter up, I found that it is the habit of parent catfishes to travel about in company with their young. Whether rightly or wrongly—and I have since failed to verify the reference—I also derived the impression that when the young aggregate in a mass, the parent occupies the center of the ball. At any rate, the supposition I framed at the time was that the peculiar marshaling of the young must depend on some form of chemo-reception. They presumably kept station by remaining within the sphere of some chemical substance emanating from the parent.

On reconsidering the matter later, a more feasible explanation seemed to be that they keep their formation in response to some

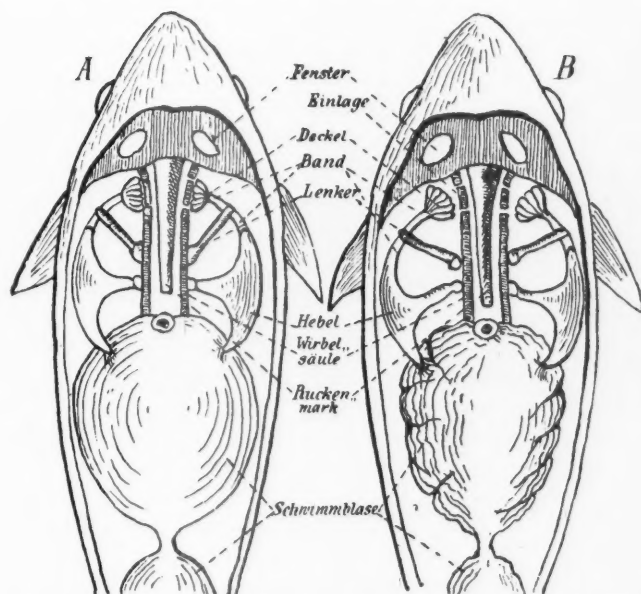


Fig. 6. Diagrammatic illustration in support of the conception that the Weberian ossicles serve as a mechanical linkage to transmit to the labyrinth simple volume changes of the air-bladder, as when its wall becomes unduly stretched (*A*) or slackened (*B*). (From Widersheim after Thilo.)

The fish exhibited is the carp. Dorsal view. Hebel = malleus; Lenker = incus; Dekel = stapes. The vertebral column (Wirbelsäule) has been split open and a part of the spinal cord (Rückenmark) left in situ.

steadily emitted central sound. Apart from the known ability of the catfish to produce sound, it is the globular configuration of the cluster of young that gives support to the latter supposition. If some chemical substance were being dispersed into the surrounding water by a moving parent, the young, instead of forming a compact van and a compact rear, would tend rather to tail behind in a long string.

For actual proof, however, one must succeed in hearing the stipulated sound. Last summer, through the kindness of Mr. B. W. Taylor, provincial biologist of Quebec Province, I received in our laboratory a pair of adult catfishes with their brood of

young. Whereas they survived for long weeks and became apparently accustomed to their surroundings, the young fish, even when the family was kept in a deep-water tank, never to my observation aggregated into a ball. Throughout the period of confinement, too, the parents emitted no sound that I could detect with the hydrophone. This case is merely one illustration of the difficulty encountered in this special type of work of dealing under laboratory conditions with events that would be more pertinently studied in the field.

I shall not here attempt to deal with the numerous experiments that have been carried out on the sense of hearing in fishes generally. Instead, I will select only three records from the literature as being of particular interest in the present connection. Maier (1909), who had sought with wholly negative results to elicit reactions to under-water sound in a large variety of different fishes, made the surprising discovery that the American siluroid, *Amiurus nebulosus*, responded to simple whistling in the vicinity of its aquarium. Both Haempel (1911) and Parker and Van Heusen (1917) by ablation experiments succeeded in locating the final receptor region for this response in the ear of *Amiurus*. There is no doubt, therefore, that some cypriniform fishes are sensitive to water-conveyed sounds, even of low intensity, and that the ultimate receptor apparatus for the purpose is labyrinthine.

So far as we have gone, we have found reason for believing that while many water-immersed animals may be sensitive to general vibration, some of them have discovered how to utilize the properties of the surrounding medium in order to communicate one with another. When fishes thus signal they use for the most part the air-bladder. Comparative anatomists tell us that the pharynx-connected air-bladder of fishes eventually gave rise to the lungs of terrestrial derivatives. The generally accredited supposition is that the fish ancestors of the tetrapod vertebrates had characteristics like those of the existing group of the Dipnoi or Lung-fishes, which, as you know, already use their air bladder as an accessory respiratory organ. We should now, however, be inclined to surmise that at least two circumstances contributed to this particular transformation, the second being that the air-bladder of the hypothetical fish ancestors was already in use and

simply remained in use as an apparatus of voice. In reality there are two things to explain: first, the adaptation of the air bladder to pulmonary respiratory use; second, the all-prevailing employment of the pulmonary apparatus as a source of voice among the tetrapods.

According to some authorities, the eventual acquisition of vocal powers by higher vertebrates was merely an incidental matter. By way of illustration may I quote the following passage from a contemporary medical writer? "The organs that make the human voice were never built for that purpose in the first place. Unlike the eye and the ear, nature built no special organ for the voice alone, but simply utilized the wind pipe and lung bellows, the swallowing parts of the food passages (tongue, lips and palate) and the nose for that purpose, long after they had taken their own particular shapes for their own special ends." As professional medical men, dealing with the whole range of ear, nose, throat and larynx, it may be some crumb of comfort to you to realize that nature, in framing these parts, went to work rather differently. Our voice and vocal mechanism have a longer lineage and more interesting history than that.

I now come to a more involved and more difficult part of this exposition. May I begin by briefly explaining some of the experimental complications that have confronted us in seeking to assign tremor or vibrational receptivity to any of the otolithic receptors of the labyrinth?

You will remember that the water vibration set up by the croaking frog can be felt with the fingers. This means that parts of the body other than the inner ear are provided with tremor receptors. Although we know from clinical observation that nerve impulses due to vibration are conveyed in the cord by their own special ascending pathway from limbs and body, we are still ignorant as to the nature of the cutaneous or other extra-labyrinthine receptors that subserve this sense. If I were to hazard a guess, I should say that they are the Pacinian corpuscles. This surmise rests merely on their site of more abundant distribution, as, for example, in the finger tips, in periosteum and in interosseous membranes, and in the mesentery of all members of the cat fam-

ily, which, as we know, have the curious habit of purring. However this may be, it is certain that tremor receptors are by no means confined to the inner ear. A delabyrinthized animal is perfectly responsive to body tremor.

In order to test responses to tremor before and after ablation of particular labyrinthine receptors, Dr. McNally and I constructed special platforms that could be set in vibration by turning a ratchet wheel. A significant thing is that most tetrapods on being vibrated give but little sign of muscular reaction. In general it may be said, however, that if the posture has previously been asymmetrical the vibrated animal immediately adopts a symmetrical pose, suggestive of "awareness" or readiness to meet the next eventuality. Otherwise it stays still. There is thus, as a rule, no very specific or characteristic reaction to mechanically conveyed tremor. This has proved an outstanding difficulty in dealing with the animal so favored by Dr. McNally for differential labyrinthine receptor ablations, viz., the frog.

Nevertheless, in some animals the reaction on a vibrating platform may be more distinct. Some pigeons, for example, may by tremor stimulation be thrown into a position of readiness for flight—why some exceptional specimens should thus behave differently from others is hard to say. In addition to inducing body symmetry, one single nick of the ratchet may, in the case of these unduly responsive animals, cause the wings suddenly to lift, the extent of the lift depending on the sharpness of the click. In their case, too, continued vibration, without actually causing flight, holds the wings steadily aloft and keeps the toes extended or unclasped. While these (infrequent) responses have an obvious bearing on the natural history of birds—on their tree-perching and perhaps also on their nest-lining habits—we have not in this connection effected any ablations of the saccule or of the lagena. One animal that gives a very conspicuous response to vibration is the rattlesnake. It rattles in response to tremor stimulation more readily than to any other form of excitation. By delabyrinthizing the rattlesnake we have been able to show conclusively that the tremor receptors involved in reflex production of tail-rattling lie, not in the labyrinth but in the middle three-fifths of the ventral part of the body—that is to say, in the body

region used by the alert or aroused animal for maintaining contact with the ground.

You will thus realize, as we soon came to realize, that even when an animal does give a visible response to communicated body tremor, it has, to begin with, a rather effective armamentarium of extra-labyrinthine receptors for the purpose. As an additional example of the kind, I might cite the cutaneous lateral line organs of fishes, which G. H. Parker (1904) found to be a tremor receptive apparatus.

Apart from this experimental complication, may I further remind you of the fact that the posterior or nonequibrial part of the labyrinth of lower vertebrates contains no fewer than four different receptor organs, the saccular macula, the lagena, the pars neglecta and the pars basilaris. In regard to the function of the two last named, I am not aware that anyone as yet has offered even a suggestion. The theoretical reasoning that led us to attribute possible auditory or tremor receptive function to the otolith-bearing lagena and saccule has acted in the direction of confining our attention for the time being mainly to these.

The mode of procedure that commended itself to us was to signal by some electrical means the responses of the eighth nerve of fishes to water-transmitted sound before and after ablation of either of these otolithic receptors. Piper (1906, 1910), who was not, however, concerned with ablation experiments, had found by electrical means that the ear of the pike (*Esox*) is responsive to water-conveyed sound. G. H. Parker (1910), by pinning down the saccular otoliths in another fish (*Cynoscion*) had been able to abolish particular body reactions to sound. Using the string galvanometer as a means of electrical record, Mr. D. A. Ross undertook to examine the responses of the eighth nerve of three different fishes, two of which, *Amiurus* (catfish) and *Catastomus* (sucker), belong to the cypriniform group, the third being the same animal as Piper originally used, namely, the pike.

Reference to the great atlas of Retzius (1881) had shown us that whereas in most fishes the lagena otolith tends to be smaller and lighter than the saccular, in cypriniform fishes this relation is reversed. In these latter forms the lagena otolith is unusually developed, while the saccular ear-stone is tiny by comparison.

On the supposition that one of the two corresponding sense organs might be for reception rather of noise and the other for reception of tone—i. e., of some particular limited range of pitch, we were inclined to imagine that the lagena, so well developed in the forms with a sounding air bladder, might, in the case of the cypriniform fishes at least, be more particularly connected with tone reception.

Unfortunately the anatomic relationships of the relevant parts prevented a proper examination of this conjecture. For adequate exposure of the eighth nerve with concomitant ablation of one or other otolithic receptor, it was invariably necessary to sacrifice the air bladder with its double chain of ossicles. Thus the surviving head parts of the cypriniform fishes could be tested for sound reception only under conditions analogous to those present in Piper's experiments on the pike. In other words, the sound, or under-water tremor, had in every case to be communicated directly to the partially immersed, surviving head, without any opportunity of being transmitted to the labyrinth by way of the air bladder and Weberian ossicles. Notwithstanding these experimental complications, Mr. Ross obtained results which prove that both the lagena and the saccular macula of fishes are concerned with sound reception, though the precise partition of duty between the two receptors is still an unsolved question.

Using as his means of producing sound a Klein pipe whose tone could be set at will at any range varying from 50 to 130 cycles per second, Mr. Ross first confirmed the work of Piper on the pike. The under-water tone, picked up by the ear, gave rise to a characteristic deflection of the galvanometer string when appropriately connected with the eighth nerve. His next step was to do something which Piper had not attempted, namely, to remove the saccular otolith and to destroy the saccular macula. This operation of itself sufficed to abolish the characteristic deflection, though there always remained some doubt as to whether the lagena had simultaneously been disturbed.

The cypriniform fish chiefly employed for the corresponding experiments was *Catostomus*. Although the deflections obtained in a similar manner with this fish were not so large as in the case of the pike, they were nevertheless characteristic and regular. In

this case removal of the lagena was invariably necessary to abolish all response. Removal of the saccular macula by itself would reduce the amplitude of the deflection, but it was plain that the lagena likewise participated in giving the full electrical effect. Thus, as I have already said, we may take it as proven that both the saccular macula and the lagena of fishes are auditory receptors.

In order to draw these remarks to some provisional conclusion, may I end by indicating what, in my opinion, is the most likely function of the saccular macula in mammals and in man?

We must remember that the cochlea and its accessory apparatus (*membrana tympani*, ossicles, etc.) are designed to vibrate in an immobile head; further, that the mere construction of the saccular receptor suggests that its stimulation involves an actual vibration or tremor of the head. We have no evidence that in any terrestrial vertebrate the saccular macula is commonly stimulated by tremor communicated to the body itself from the outside—that is to say, we have no evidence that the saccular macula of land vertebrates is a simple exteroceptor. When we consider that all production of voice by air breathing vertebrates is liable to cause head tremor, it is perhaps a fair inference that the saccular macula in these animals is a proprioceptor involved in the emission and regulation of voice.

This would mean that we hear our own voice with the help of two kinds of receptor, while we hear the voice of our neighbors with only one. If this were so, it would be easy to understand why we fail to recognize a phonograph record of our own voice as easily as it is recognized by our friends. The suggestion here advanced is, of course, open to investigation by the recognized method of comparing careful clinical records with subsequent postmortem findings. Occasional cases may arise in which the saccular apparatus may be thrown out of commission without involving the cochlea. A more common type of case is probably one in which cochlea disability is grave while the saccular maculae remain unaffected. Only when a number of properly selected patients have been thoroughly examined and interrogated from this point of view and the findings subsequently checked by anatomic examination, shall we be able to say whether the hearing of

our own spoken voice involves the mediation of two separate labyrinthine receptors, cochlea and saccular macula.

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MUCOSAL IMMUNITY IN THE NOSE AND
ACCESSORY SINUSES.*

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For two generations we have been following paths blazed out for us by illustrious explorers in medicine, always looking back a little wistfully to be sure that we have followed the right trail. Of late, some hardy spirits have broken out of established ways in histopathology and immunology, and they return telling strange tales in a new tongue. Physical chemistry, with its mathematical demonstrations of elemental mutation, has supplemented the mnemonic processes of qualitative analysis. Tissue cultures, vital staining and discovery that body cells and bacteria may change shade, size and function in response to varying stimuli, are disrupting older classifications by formal anatomists, pathologists and bacteriologists.

Claude Bernard, father of modern experimental medicine, early stigmatized our tendency to cling to systems. In 1865, he said:

" . . . These theories and ideas are by no means immutable truth; we must always be ready to abandon them, to alter them or to change them as soon as they cease to represent the truth. In a word, we must alter theory to adapt it to nature, but not nature to adapt it to theory."¹

W. B. Cannon of Harvard has recently applied a concept of Claude Bernard's to his splendid studies on the sympathetic nervous system. Recalling that we are protected from the outer world by a horny layer of dead cells, and elsewhere either by a film of salt solution or mucus, he calls this film the "fluid matrix," whose composition depends largely upon the proper functioning of the involuntary, sympathetic or "interofective" nervous mechanism.²

*From the Department of Otolaryngology, University of Oregon Medical School.

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Within this fluid matrix our body cells function, not as watertight, solid-walled compartments, but as amoeboid, fluctuant vital elements, subject to perpetual change, growth, increase, death—flames burning under water, in Crile's arresting simile; an unforgettable sight in living culture, as feelingly stated last year by Mosher.³

Variables affecting the cell may (1) be extrinsic, including the chemical and serologic setup of the blood, mucus, serum and other divisions of the fluid matrix; nerve terminals and their charge of electric potential; environmental and body heat, humidity, air or gas pressure; or (2) the variables may be intrinsic, within the cell wall; the kind of cell, modified by inherited constitution, by relative proportion of nucleus to cytoplasm, by location, function, completeness of its nerve connections, ability to ingest foreign matter, power to change function, shape and size under varying stimuli, capability of regenerating and filling in wounded or defective areas, and the like.

The difficulties of studying all these factors are very great. James Gray regards "living material as a state of matter where the constituent molecules are organized in a way quite unknown in the inanimate world, and which will only be elucidated by methods of analysis which have yet to be discovered. . . . Until we have the means to explore the situation in much greater detail, a knowledge of our ignorance is perhaps the most valuable asset we can hope to possess."⁴

Within recent years, thanks to research initiated by Mosher and his pupils, we have learned that accessory sinus membranes tend to regenerate ciliated epithelium, when proper conditions for healing occur.

Mosher and Ross' work on tissue cultures from nasal mucosa has demonstrated the necessity of embryonal tissue extract as an aid to artificial growth.⁵ Hilding, complementing Mosher, Knowlton and McGregor's studies in regeneration, has demonstrated the necessity of vascular granulation tissue as a proper soil for such regeneration. Where poor vascularity exists, fibrous changes are rapid, and flat scars and bands or diaphragms may form, without regrowth of ciliated epithelium. Further, Hilding has demonstrated a definite change in character of the sinus mucosa of

dogs, with exposure to open air.⁶ Thin ciliated mucosa becomes cuboidal and squamous when thus laid bare. In rabbits, when one nostril is permanently occluded, ciliated mucosa becomes much thinned and the ciliated cells are almost all replaced by goblet cells containing mucin.

These changes in epithelium, reflected in the overly roomy nose after turbinectomy and in ozena, or in cystic degeneration of antral walls after long closure of the ostium, depend upon changes in function, exposure to colds, irritants and the like. Interference with the moving network of mucin, which changes every ten or fifteen minutes on the active turbinal areas of ciliary action, breaks the protective fluid matrix and permits irritants to reach the cells.⁷ The rapidity of desquamation and replacement of epithelial cells is astounding, which leads us to the thought that epithelial structures are not the essential in mucosal defense.⁸ Instead, we are led to consider the underlying connective tissue as the important barrier to invasion of the body through these areas. It should also be remembered that absorption through the sinus mucous membranes is relatively slight, and opinion now tends to minimize the sinuses as sources of focal infection. Rather they may act as reservoirs of infective material which passes down into the respiratory and digestive tracts and is elsewhere absorbed.

The connective tissue layer of sinus mucoperiosteum increases greatly in thickness with irritation.⁹ This thickening is due to increased vascular activity, and the mobilization of histiocytes or phagocytic connective tissue cells, some of which are latent components of the membrane at all times, while others come in by diapedesis from the capillaries. Such cells are normally frequent in granulation tissue, and actively take up the work of disposing of foreign matter or bacteria penetrating the ciliated epithelium. After this work is done, they change into fibrocytes and help build up solid reparative tissue. Depending upon the nature and severity of the irritant, they are aided by polymorphonuclear leucocytes and lymphocytes, from the blood stream.

Eosinophile leucocytes are specifically attracted to the tissues by certain protein stimuli, notably allergens from plant and animal matter, including the toxic emanations of various animal

parasites. Leucocytes seem to accompany the stage of active repair, following after the early histiocyte mobilization. It is noteworthy that tonsil tissue cultures are definitely toxic to histiocytic and fibrous elements;¹⁰ and histiocytes are found only in the fascia about lymphoid structures, never in the stroma.¹¹ Phagocytosis by histiocytes of degenerated lymphocytes has been observed after irradiation.¹¹

We have, first, the constantly moving film of mucin; second, the ciliated epithelium; and third, the connective tissue layer with its blood supply, as active factors in mucosal immunity. Both the first and second are adversely modified by cold, trauma and by dilution of the mucous secretion.⁷ Connective tissue resistance remains a constant defensive factor here, as in the skin and other epithelial surfaces.

We have elsewhere considered local factors modifying such immunity, and shall not here anticipate the results of pending studies made through the generosity of the American Academy of Ophthalmology and Otolaryngology. Nevertheless, it seems proper to suggest certain applications of the connective tissue defense theory.

Corbus has recently secured rather startling results against gonorrhoeal infections by intradermal immunization with a gonococcus bouillon filtrate.¹² It is necessary to reach the true skin or mesodermal layer to secure histiocyte stimulation through the sympathetic nervous system, in his opinion. Here is a suggestion for the employment of upper respiratory vaccines—intradermally instead of subcutaneously. The so-called "Arthus phenomenon," solidification of tissue by repeated injection in the same locality, may cause necrosis and must be guarded against. Corbus suggests intradermal injections at least six inches apart.

Mosher's work with Ross in tissue culture suggests the desirability of an extract of embryonal tissue, for local use in improving healing, or perhaps by intravenous or intramuscular injection. Such a tissue is packed with stellate connective tissue (embryonal) cells, which correspond to the histiocytes found in profusion in the omentum.

In the abdomen, they are concerned with rapid emergency defense measures, and set up fibrous barriers very promptly. Charl-

ton has extracted an alcohol-soluble substance which he calls "reticulin," from the omentum of young pigs; this is reported as singularly active in hastening reparative processes. Whether the "growth hormone" or "alexin" recently reported from London partakes of this character is as yet not settled.

At all events, we may hope to see the functions of the sympathetic nervous system, notably in its sphenopalatine ganglion and the nervus terminalis,¹⁴ reduced to effective control within the next few years. Until we secure this method of local regulation of immune reactions, we are justified in making use of such toxins and proteins as are specific to the individual, and perhaps of preparations made from embryonic connective tissue, by the method of intradermal stimulation. These are powerful agents, and dosage must be minimal, increased very slowly. May we not bespeak attention on the part of those enabled to carry on clinical and laboratory research to the necessity for exhaustive work along this line, as well as in the equally important field of cellular physiology as related to diet and chemical equilibrium within the mucosa? Connective tissue is, fortunately, resistant to toxins and other external stimuli; it equally resists all manner of local attack by drugs. Here we must turn to the researches of cellular physiologists like Gray and Gellhorn, having regard to the extraordinary activity of the hydrogen ion in cellular function^{15 16} and to the necessity of regulating mechanisms within the cell which maintain the natural reaction. Alterations in osmotic tension affect permeability of the cell walls,^{17 18} and differences arise not only from the reactions of the nutrient medium in which the cell is bathed, from its content of various salts, but also from the circulating "incretions"—substances carried to the cell from the glands of internal secretion, as activated by the sympathetic nervous system.¹⁹ Upon these facts rest the important dietetic work of Stucky, Dean and Shurly, and the metabolic attack advocated by Jarvis and Lewis.

Behind and beyond all these factors in mucosal immunity lies the original setup of mesenchymal tissue which comes to each individual by inheritance. Constitutional predisposition, for which as yet there are few exact criteria from the histopathologists, though plenty of data exist in anthropometric and actuarial sta-

tistics, will be found to play an important, often a decisive rôle in settling the question of individual immunity to respiratory disease.

May I remind you that Wagner discussed the natural immunity of the mucous membrane of the respiratory tract before this association thirty-four years ago?²⁰ Since that day, surgical and bacteriologic technic has reached extreme perfection, and now we return to the mystery of life itself, facing again the question of natural immunity as based on the vital resistance of connective tissue. In such study we should have always before us these words of Claude Bernard:²¹ "We are still very far from this absolute truth; and it is probable, especially in the biologic sciences, that it will never be given us to see it in its nakedness. But this need not discourage us, for we are constantly nearing it; and moreover, with the help of our experiments, we grasp relations between phenomena which, though partial and relative, allow us more and more to extend our power over nature."

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LVI.

THE USE OF VARIOUS TYPES OF AUDIOMETERS IN
CLINICAL WORK.*

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Audiometric studies which have appeared in the literature have dealt chiefly with specific research problems. In fact, many have considered the audiometer to be distinctly a research instrument. This is probably a natural consequence since it was developed in an attempt to solve a research problem. It does not necessarily follow that the application of the instrument must be confined to the fields of research. The history of medicine contains records of many devices which early were thought to have little clinical application but were later accepted and have become routine; and usefulness is the one standard which determines whether or not a method or device shall be accepted.

Several types of audiometers have been installed in the Otollogical Clinic of Washington University Medical School during the past two years, and audiometric tests are being made routinely on all ear cases presenting themselves at the clinic. Such routine can scarcely be called research. The tests are not conducted by a single person. Each junior member of the clinical staff who first sees these patients receives a course of instruction in the use of tuning forks, audiometers and other devices used in the functional tests of hearing, in the technic of testing and in the interpretation of the results. He then makes a complete examination of his patient and presents his record to a senior staff member who criticises it and assists him in building up the clinical picture and aids him in deciding the proper method of treatment.

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From the Department of Oto-Laryngology: Oscar Johnson Institute, Washington University Medical School. The Ball Fund.

This procedure has a twofold purpose. First, it insures a complete and careful examination and analysis of the results in each case; and second, the junior who has completed his period of training is capable of judging what he considers essential in his own work when he assumes his practice.

The interpretation of the results of the functional tests of hearing depend upon a comparison of the result secured with a standard of normalcy. During the two years in which these tests have been conducted, the quarters assigned for this purpose have been changed several times. Early in the period no soundproof room was available. It has also been necessary to take the apparatus to a hospital room or ward and conduct the tests under quite unfavorable conditions because the patient could not be moved from his bed. The use of several different types of audiometers under these varied conditions by at least fifteen different examiners adds several complications to the establishment of normals. This is perhaps best illustrated by tests under varied conditions on a single individual. It is obviously impossible to work out averages on a large number of individuals under each of these conditions.

Fig. 1 shows four records secured with three types of audiometers from tests made in different rooms on one of our younger laboratory assistants. The audiogram lettered *A* was taken in a soundproof room using the 2A audiometer. It will be noted that the curves for both ears lie close together on the chart and both show much better hearing than the printed normal. The audiogram lettered *B* was taken with the same instrument in one of the laboratories which was fairly quiet at the time but not soundproof. The curves in this more nearly follow the printed normal. The audiogram lettered *C* was made in the soundproof room using the 1A audiometer. Here again we see considerable variation from the printed normal. The chart lettered *D* shows the result when the same person was tested with the 4A (phonographic) audiometer. The first three of these records show the futility of comparing records with standards made under other conditions. For our work, the charts offer simply a convenient method for plotting our results, and the interpretations must be on the basis of our standard conditions. It is not assumed that this person had

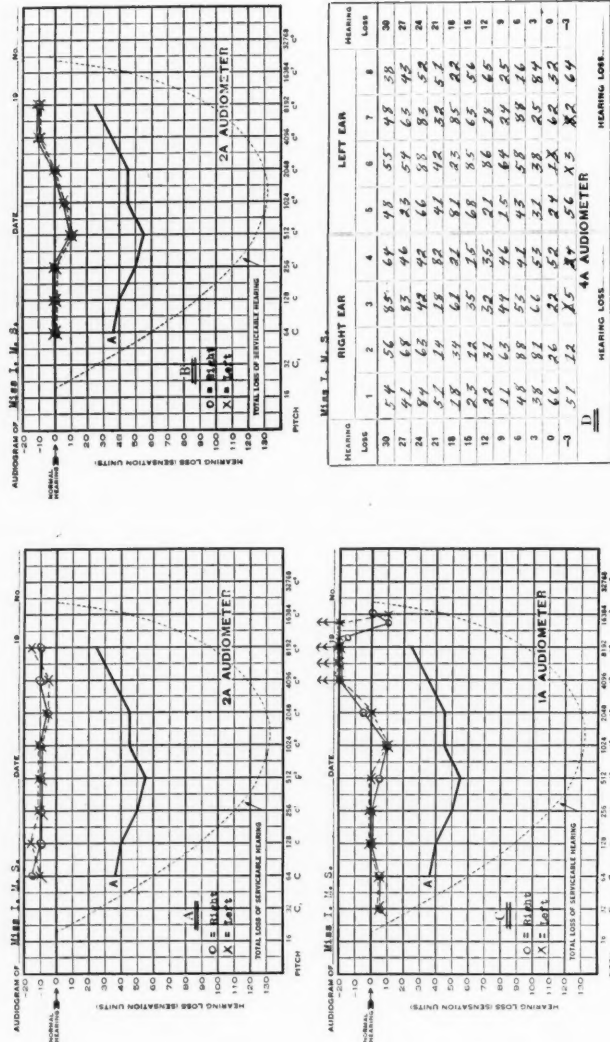


Fig. 1. Audiograms of a young adult with normal hearing made under varying conditions with three different types of audiometers.

exceptionally acute hearing. These records could have been duplicated by those on several other workers from the laboratories.

The record secured with the 4A audiometer, although made in a soundproof room, was not made under soundproof conditions. The grinding noise of the phonograph motor added a masking error which can only be eliminated by constructing a soundproof box for the phonograph. It has been considered unnecessary to do this for this particular type of test.

A tentative normal for the conditions of our tests once established, all results, no matter by whom the examinations are made, are compared with this standard. Each one doing the tests is instructed as to the significance of this normal and his interpretations are based upon it.

One criterion for judging the usefulness of tests of this kind is that it gives direct information as to the progress of a condition which is undergoing treatment. Fig. 2 illustrates three records showing the changes in hearing occurring in a period of two years in a case of otitis media. The curves given at the first test are shown in the two charts by the connected circles. At this time only the left ear was affected, the curve for the right being close to our tentative normal. A myringotomy was done on the left and the trouble apparently subsided. In February, 1931, the ear condition recurred, this time involving both sides. Both drum membranes were incised and the patient had very little trouble until the following November. At this time she was admitted to the hospital and the simple mastoid operation was done on each side. On January 19th, the acuity of hearing is indicated by the curves joining the rectangles and the record of a test made on February 16th by the curves of connected crosses. The hearing power remains somewhat diminished and the patient is still under observation.

Fig. 3 shows four records in another case of otitis media. The acuity on November 9th, when only the right ear was involved, is shown by the curves joining the circles. The record of January 26th, shown by the curves of connected crosses, indicates the maximum involvement. Successive tests show on both sides a slight but gradual improvement, and according to notes from the clinical charts, this was the chief evidence for delaying operative

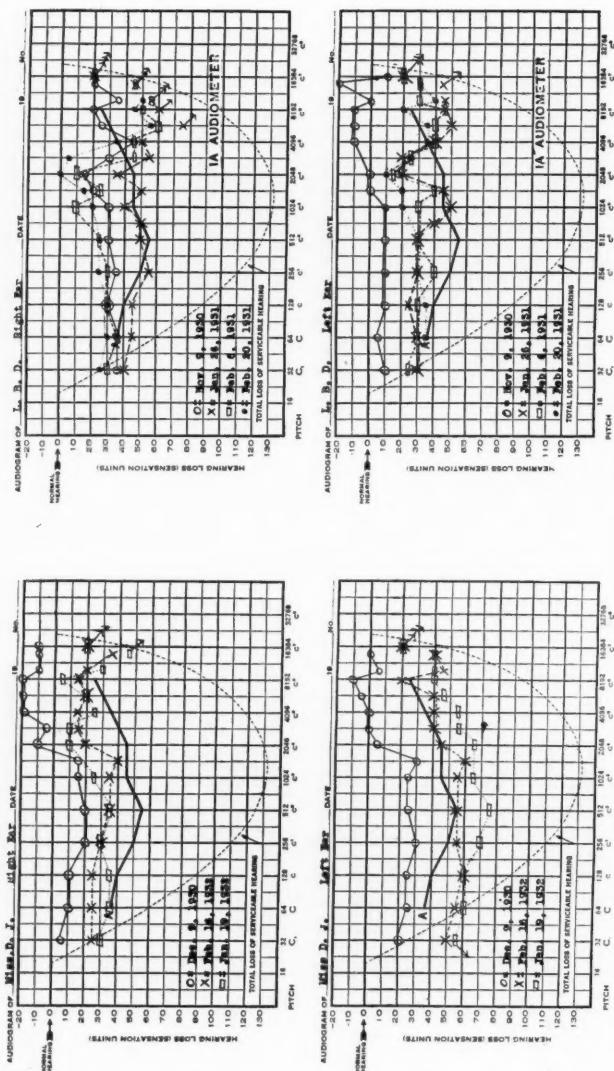


Fig. 2. Audiograms showing the progress during a two-year period in a case of bilateral suppurative otitis media.

Fig. 3. Audiograms showing the progress in a case of bilateral suppurative otitis media.

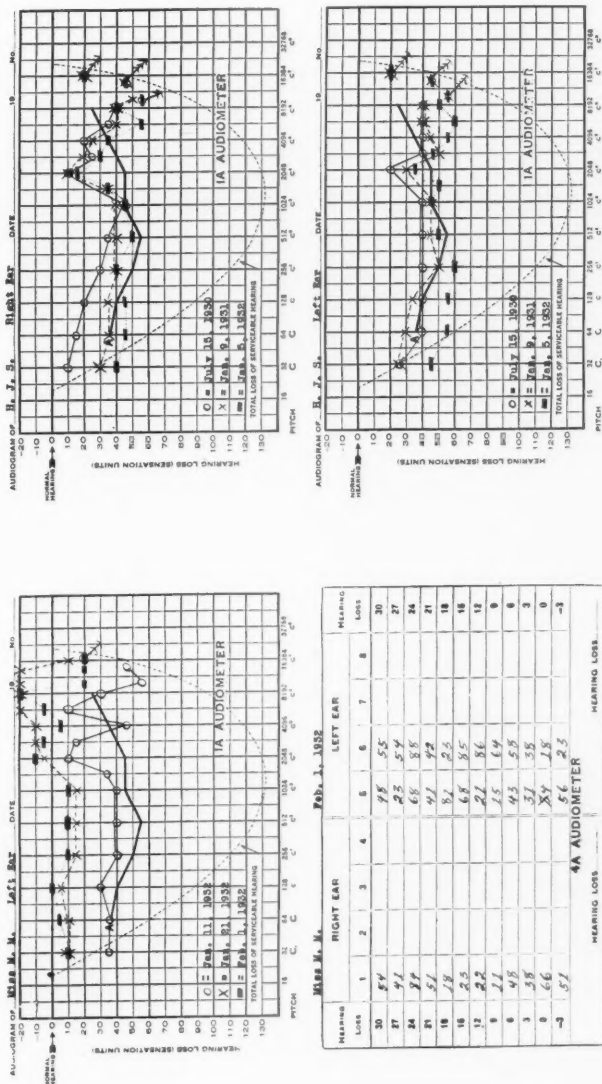


Fig. 4. Audiograms showing the progress over a period of ten days in a case of unilateral acute suppurative otitis media.

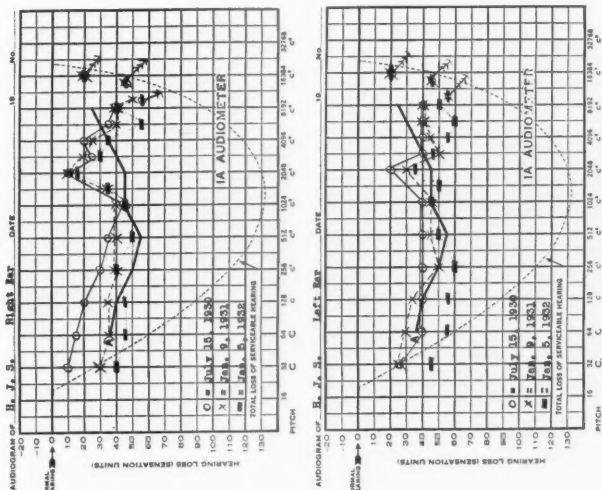


Fig. 5. Three records covering a period of eighteen months in a case of progressive deafness (non-suppurative).

work, and the patient eventually recovered without operative intervention.

Fig. 4 shows the records in a case of unilateral otitis media, the left being involved. This patient announced at the time of the first test that she was completely deaf in this ear, but the audiometer test showed only moderate loss. Under conservative treatment the condition cleared up but she felt that her hearing was still considerably diminished, and it was only by permitting her to change the receiver from her good ear to the affected one that she was convinced that her hearing had returned to normal. The record with the 4A (phonographic) audiometer demonstrates the equality of the hearing in both ears when the otitis had subsided.

Fig. 5 shows three records for the right and left ears taken during a period of eighteen months in a case of progressive deafness of a nonsuppurative type. The Rinne test was negative, bone conduction was not decreased, and there was a positive response to the stapes fixation test. The results indicate that the deafness is slowly progressing, in spite of treatment, although the patient notices no additional loss for conversational voice. The patient is a minister and with this evidence of progress of the deafness is attempting to make the necessary adjustments to enable him to carry on his occupation.

Fig. 6 shows two records taken with an interval of a year. This patient had, previous to the time the first test was made, consulted a physician as to the condition of her ears and had followed his advice as to treatment. In such cases a single test is not sufficient to determine whether everything possible has been done. With these two records it was evident that there had been no progress in that time and both the patient and her physician were reassured.

Fig. 7 shows the records of three tests made at six month intervals on an eight-year-old patient. The child had been quite a problem to her parents. She was slow in learning to talk. It was thought that due to a known birth injury something might have happened which was responsible for her slow mental development. The three records lie as close together as one could expect in testing a child of this age, and it is evident that the deafness has not progressed during this time. The striking thing brought out

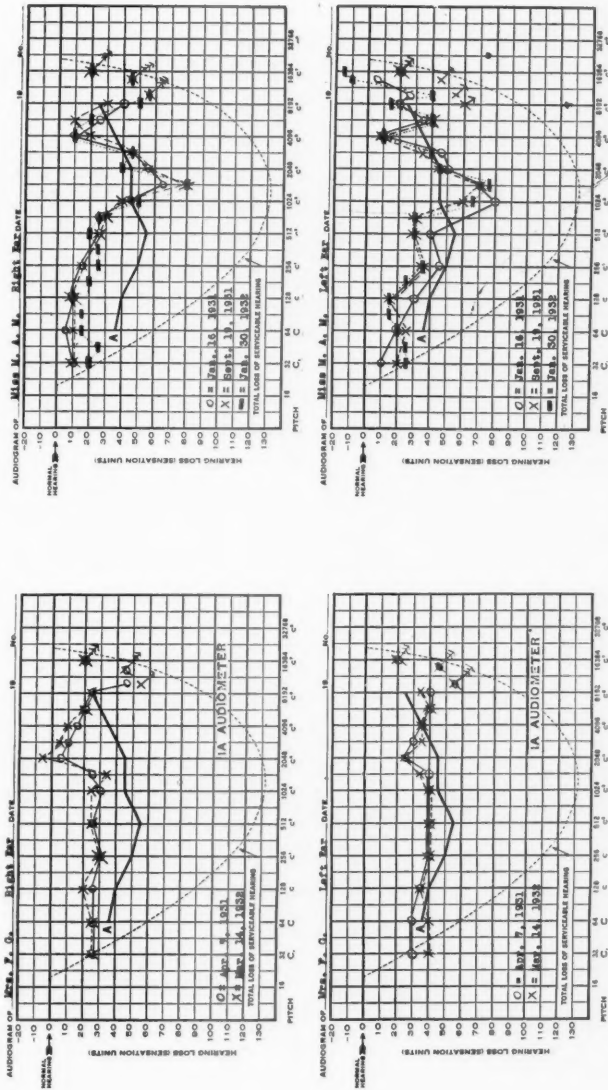
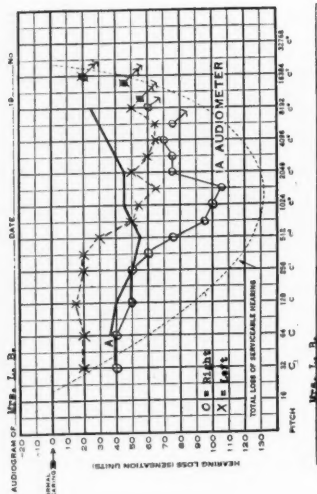
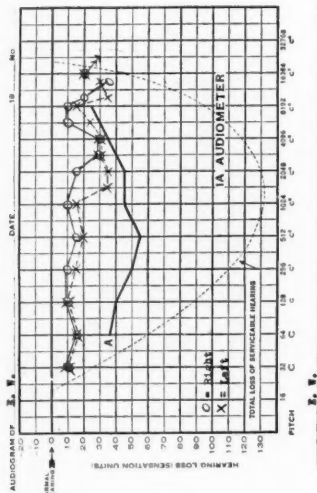


Fig. 6. Two records taken with an interval of a year showing little or no change in acuity.

Fig. 7. Three audiograms at six months intervals showing no advancement in the deafness.



RIGHT EAR		LEFT EAR	
HEARING	LOSS	HEARING	LOSS
30	54	54	55
27	41	41	52
24	27	44	42
21	21	44	31
18	18	44	18
15	15	44	15
12	12	44	12
9	9	44	9
6	6	44	6
3	3	44	3
0	0	44	0
-3	-3	44	-3



RIGHT EAR		LEFT EAR	
HEARING	LOSS	HEARING	LOSS
30	54	54	55
27	41	41	52
24	27	44	42
21	21	44	31
18	18	44	18
15	15	44	15
12	12	44	12
9	9	44	9
6	6	44	6
3	3	44	3
0	0	44	0
-3	-3	44	-3

Fig. 9. Two records illustrating the correlation in results secured by tests with the 1A and 4A audiometers.

Fig. 8. Comparison of the results secured from tests with the 1A and 4A audiometers.

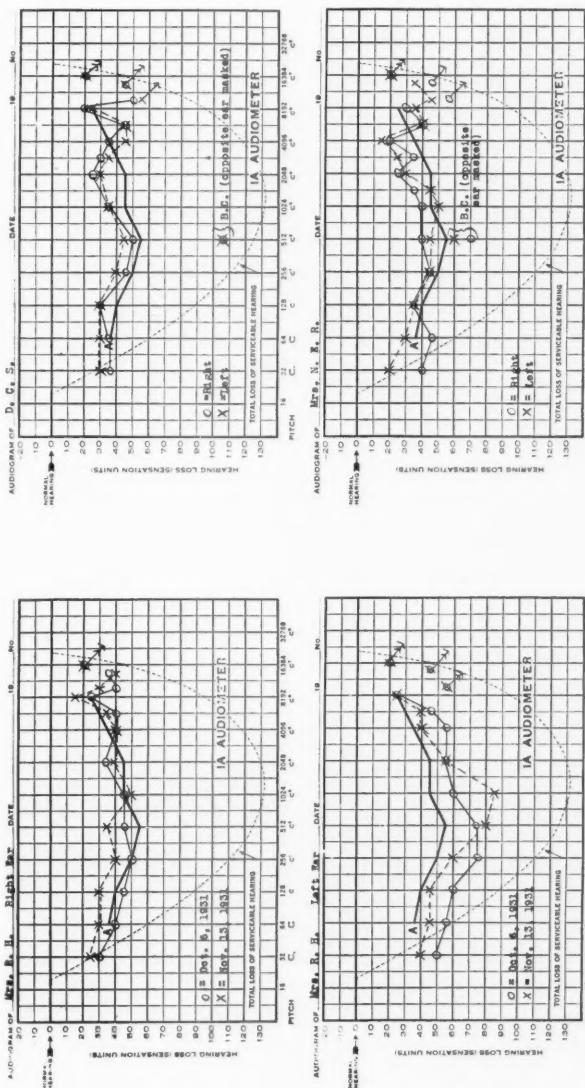
by the examination was the marked loss in acuity for tones in the range near 1024 d.v. This marked loss, coming almost in the center of the range covered by the spoken voice, might easily account for the retardation in learning to speak, for what she hears from conversation is so different from that heard by normals it seems rather a wonder that she did eventually learn to speak very well.

Fig. 8 shows the records from tests made with the 1A and 4A audiometers on a man fifty-six years of age. The record from the 1A audiometer shows essentially normal hearing in both ears for tones below 1024 d.v., with a marked decrease in acuity above this pitch. The record with the 4A audiometer shows that with the right ear he heard correctly four words out of six when the intensity was three units fainter than the normal, but he began missing words when the intensity was fifteen units louder than the normal. The left ear which shows a greater loss at 2048 d.v., gave a much poorer record, words being missed when they were twenty-four units louder than normal, and he was able to get only one word correct when the intensity was three units louder than the normal.

The last two figures are illustrative of a large group who misunderstand spoken words because of their loss in acuity for the high overtones which carry the distinguishing characteristics of vowel and consonant sounds.

Fig. 9 shows the records of a case where this condition is much more marked. The tones in the lower part of the speech range—i. e., 256 d.v. and 362 d.v., are heard in the left ear, according to the record with the 1A, with about five units loss, but 1024 d.v. and 2048 d.v. show a much greater loss. The record for the 4A audiometer verified this, for mistakes in the left appear at twenty-seven units loss. The right ear shows forty or more units loss for all tones and the 4A audiometer with its maximum of 30 units was inaudible.

Fig. 10 illustrates more clearly the limitations of the 4A audiometer. Both records show essentially normal hearing in the right ear. The curve for the left more or less closely parallels that for the right in the record secured with the A1 audiometer but lies from twenty to forty units lower on the chart. Consequently the words from the 4A are inaudible in the left ear. Deafness of this degree is frequently encountered in clinical work, and the 4A



audiometer would have a greater clinical application if it covered a wider range of intensity values.

Fig. 11 illustrates again the correlation between the results secured with the two types of audiometers.

Considerable interest has recently been shown in the effects of thyroid treatment on certain types of deafness. The only method of determining the change accompanying such treatment is by actual tests. The case whose record is shown in Fig. 12 had negative responses to the Rinne test on each side, bone conduction was not decreased, and had positive responses for fixation of the heads of the stapes. The first record was made (October 6th) before the treatment was instituted and the second after about a month of thyroid treatment. She thought her hearing was much improved at the time of the second test. Low tones were heard in both ears, but the acuity for high tones remained practically unchanged. It has not been possible to determine the final result of the treatment in this case.

Some persons have contended that one should be able to make a diagnosis of ear conditions on the basis of the information given in the audiogram. Fig. 13 shows the fallacy of this assumption. The record at the top of the chart was secured from tests on a man, 68 years of age, and the lower from a woman of 45 years. Bone conduction was markedly decreased in the former and normal in the latter. The man gave a positive response to the Rinne test and the woman a negative. The man had noted his deafness for about six months, but the woman had been hard of hearing for eleven years. The upper limit for bone conduction with the monochord was 14,000 d.v. for the man and 18,000 d.v. for the woman. The man entered the hospital for treatment of a suspected brain tumor and the woman because of a breast tumor. The audiograms are similar in their general contour but other clinical tests make it evident that the pathology in the two cases is quite different.

The patient whose record is shown in Fig. 14 was one of a group of probationers entering the training school for nurses. A routine test with the 4A audiometer showed some loss of hearing in the left ear. She returned for a more complete examination and in the tests with the 1A audiometer gave the results at

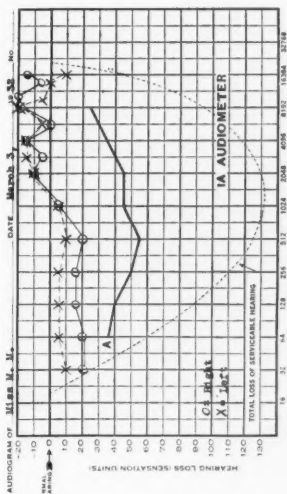
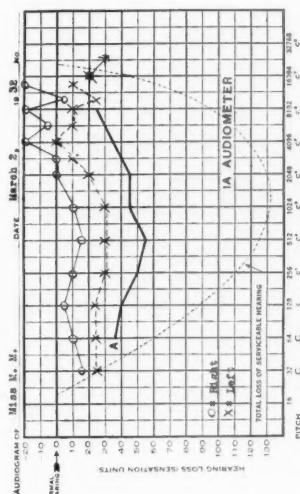
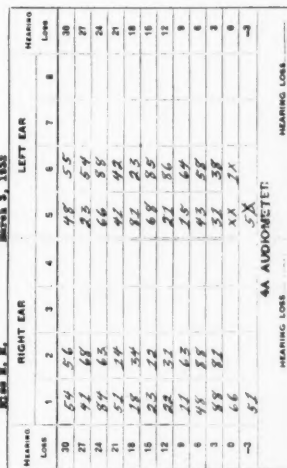
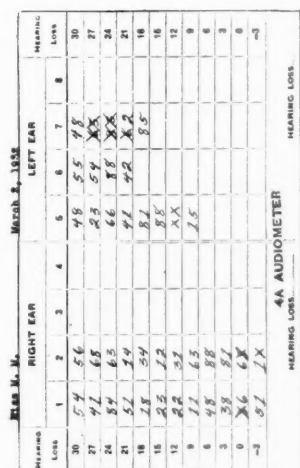


Fig. 14. Audiograms taken before and after the removal of impacted cerumen.

the top of the chart. Otoscopic examination revealed the presence of a large plug of cerumen in each ear. That in the left was removed with considerable difficulty. Tests after the removal of the cerumen gave the results shown in the two records at the bottom of the chart.

SUMMARY.

The records shown have been selected to illustrate some of the practical applications of audiometers to clinical work. The results secured in the audiometric tests have been shown to give information not only to the patient but to the clinician as well, aiding him in making a diagnosis and helping him to determine the efficacy of the treatment he has instituted.

LVII.

CAN WE SCIENTIFICALLY ADVISE PATIENTS AS TO
THE EFFECTIVENESS OF HEARING AIDS?*

HARVEY FLETCHER, PH. D.,

NEW YORK.

This question is frequently brought to the attention of otologists because hard of hearing patients are constantly asking for advice. Generally, the advice given is to try two or three sets, and then, from the patient's experience with them, determine for himself whether any of them is of service or not. What would you think of an ophthalmologist who would advise a patient to obtain glasses in a similar manner? Of course, I realize there is not a strict analogy between the two cases, but I am sure you will agree that such advice is far from satisfactory from the patient's viewpoint.

It is my purpose in presenting this paper to give such facts concerning speech and its interpretation, types of deafness, characteristics of hearing aids, and the reactions of deafened persons, as bear upon this problem and as have emerged from our research studies at Bell Telephone Laboratories along related lines.

First let us consider the nature of speech. Its loudness varies as the speech proceeds in a manner indicated in Fig. 1. This shows the variations in loudness level as the sentence "Joe took father's shoe bench out" is spoken, the time scale being represented on the horizontal axis. If the ear of a listener who has normal hearing is one meter away from the mouth of the speaker, he will experience changes in loudness about as indicated on the scale under the caption "Normal Hearing." The numbers used are decibels or sensation units above the threshold of hearing. The scales at the left of this normal are for persons having the hearing loss as designated at the top of each column. For comparison purposes, the dots at the bottom indicate the average noise level in which conversation is usually carried on. Of course, this varies a great deal but it represents a typical condition.

*Read before the annual meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., May 23-25, 1932.

For example, for a person with normal hearing the variations of the level of the speech cover a range of 40 to 70 db above the threshold of hearing, the faintest speech sounds being about 10 db above the noise level. A person having a uniform hearing loss of 30 db will therefore not be able to hear the noise but will have no difficulty understanding speech. Under these particular circumstances he is better off than a person with normal hearing. However, all persons do not speak with the same intensity. Experiments upon the intensity used in conversation of a large number of different persons showed that 10 per cent of them used intensities which are about ten db lower and 5 per cent used intensities which are six db higher than the value shown in Fig. 1. If the curve in Fig. 1 is lowered 10 db to correspond to these lower 10 per cent of speakers, it will be seen that a person having a uniform 30 db loss of hearing will still have no difficulty in interpreting conversational speech from a speaker at the distance considered.

For a person having a hearing loss of 40 db, we have a borderline case. He will be able to understand a person speaking with the normal intensity. For the lower 10 per cent, he will be unable to hear a number of the consonants. However, upon indicating his difficulty in hearing, the speaker will generally raise his voice or the two will come closer together so that for conversational purposes we are led to the general conclusion that a person having a hearing loss of 40 db or less, will not require the use of any hearing aid. Although under some circumstances he might be able to hear slightly better, these cases will be so few that they would not compensate for the inconvenience of using such a set. Our experience with a large number of hard of hearing persons has indicated that this general conclusion is correct.

It is seen from Fig. 1 that when one's hearing loss is between 50 and 60 db, one will only hear the loud vowel sounds when the speaker is one meter away, and the sentence "Joe took father's shoe bench out" will sound like *ō, u, a, r, ū, e, a, ū*. One would necessarily be very expert to make sense out of such a series of sounds. For a hearing loss of 70 or more, no sounds whatever would be heard. It is thus seen that for persons having a hearing loss greater than 40 db, either the distance between the speaker

and the listener must be considerably less than one meter or some means must be used to amplify the speech before it will be understood. Bringing the mouth of the speaker from one meter to close to the ear of the listener will increase the speech intensity about 40 db. This indicates that a person with as much as an 80 db loss of hearing can hear and understand speech when it is spoken directly into the ear. Since a person is usually embarrassed when he is required to talk at a distance from the listener smaller than about one-half meter, it is usually more advantageous to use a hearing aid for producing the amplification rather than require the speaker to come closer to the listener than this distance.

For conversational purposes, then, a person having a 50 db loss requires a set having about a 10 db amplification. In general, one having a hearing loss L requires a set having an amplification ($L-40$). If then a set were made having a varying amplification from 0 to 40, it would give satisfactory performance for cases of deafness from 40 to 80. For the more severe types of deafness, a compromise between the distance of the speaker from the microphone and the amplification of the hearing aid must be made. For example, if the hearing aid had no amplification whatever, but had sufficient capacity to carry the speech power, then if the microphone were placed close to the lips of the speaker the listener would hear the speech just as loud as though the speaker were talking directly into the ear. There are a number of sets on the market which are approximately in this category and are used in this way. When the hearing loss is greater than 80 db, the capacity of the hearing aids which are usually sold is inadequate and special equipment which will handle larger amounts of power is necessary.

All that I have said has been based on the following assumptions: First, that the hearing loss is a uniform one; second, that the sound powers involved are such that they will not hurt the ear, and third, that the amplification of the hearing aid is uniform. The first is seldom true; the second is only true with moderate degrees of deafness; and the third is only true in specially designed sets. For purposes of determining what recommendations to make concerning the use of hearing aids, the four classifications shown in Fig. 2, called A, B, C and D, may be useful.

In this figure the auditory sensation area is divided into four parts by the lines 1, 2 and 3. These lines only extend two octaves on either side of the 1024 cycle tone—that is, from 256 cycles to 4096 cycles. It is this region that determines the recognizability of the speech sounds. Any audiogram lying above line (1) is classed as A; one lying between lines (1) and (2) as B; one lying between lines (2) and (3) as C; one lying below line (3) as D. The position of the audiogram either on the upper or lower pitch side beyond the dividing lines does not influence the classification.

Using these classifications, we can make the following generalization concerning the use of a hearing aid for conversational purposes. Any person having a Class A audiogram will not receive sufficient help from a hearing aid to justify the inconvenience of using it. A person having a Class B audiogram is a candidate for a hearing aid. The audiograms in this class are divided into three subdivisions called 1-B, 2-B or 3-B, according to whether the audiogram is approximately horizontal, sloping downward or sloping upward. Cases in Class 1-B will be most easily satisfied with a hearing aid. Those in Class 2-B will require hearing aids which are much more carefully designed in order to obtain the same degree of satisfaction. Such cases cannot tolerate as much distortion as those in Class 1-B. As you know, such cases are usually due to nerve deafness and many otologists tell such patients that a hearing aid will not help them. Our experience has shown that they can be helped if the set being used gives very good quality of reproduced speech. Cases in Class 3-B occur only very infrequently but they may be treated the same as Class 1-B. For purposes of illustration, consider the audiograms in Fig. 3. Cases 1 and 2 are Class A. Cases 3, 4, 6 and 7 Class 1-B; Cases 8, 9 and 10 Class 2-B and Case 5 Class 3-B.

The cases falling into Class C require more powerful aids, which at the present time can only be produced by using vacuum tube equipment. Such sets are usually bulky and expensive. However, they are a great boon to those who badly need them. Two such cases whose audiograms are shown in Fig. 4 are now successfully using such equipment. They are able to converse without much repetition by using such amplifying

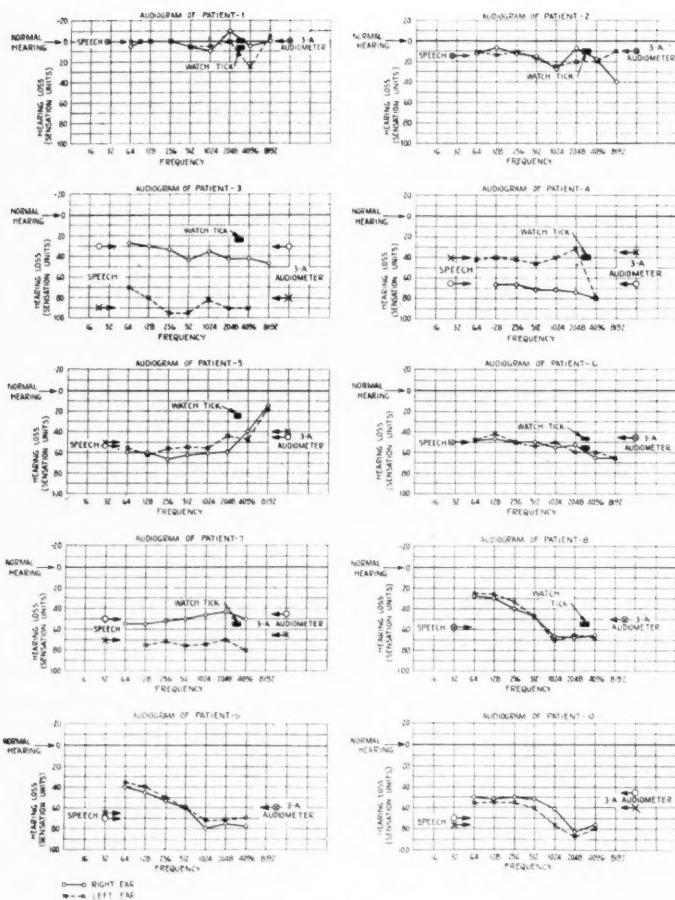


Fig. 3.

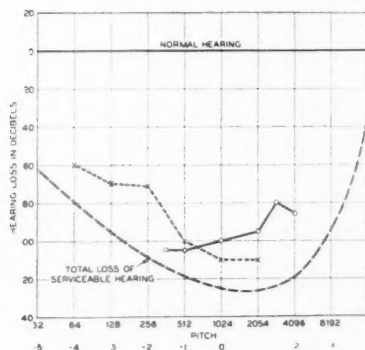


Fig. 4.

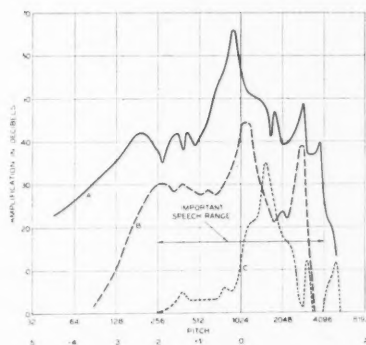


Fig. 5.

equipment and can hear nothing at all, even at fairly close range, without it. Those in Class D, according to our experience, cannot be helped by any equipment now available. The vowel sounds hurt them before the consonant sounds are above their threshold.

Although it is desirable in most cases for a hearing aid to have a uniform amplification, this is never realized, even approximately, in commercial sets sold in the open market. To illustrate this, the amplification A expressed in decibels for three types of set now being used is shown in Fig. 5.

To determine the effectiveness of any set for conversational purposes for persons whose hearing loss is known, the following procedure is suggested. This procedure is based upon our re-

search work on the effect of various kinds of distortion upon the ability of the average person with normal hearing to interpret speech.

There are two principal reasons why one fails to recognize a speech sound, namely, (1) the sound is not loud enough to be well above the threshold of hearing, (2) the relative intensity of the components of the sound has been changed in the process of transmission. Experiments upon the rôle played by each of these factors have shown that for a hearing device a figure of merit M which is related to the recognizability of the reproduced speech can be calculated by a formula of the type

$$M = \int W.D.dP \quad (1)$$

where W is a function which depends only upon the amplification A of the set at each pitch P and D only upon the pitch. The mathematical procedure indicated by this equation can be reduced to a very simple operation which is sufficiently accurate for most purposes. For applying this procedure to any set you must first know its amplification characteristic A , such as is shown for the three sets in Fig. 5, and also the hearing loss H for the person who is to use it. Then the following table may be used to calculate the figure of merit M . The figure of merit M is found by adding together a series of ten numbers corresponding to the ten horizontal lines in this table. From the amplification curve of the set a figure for A is obtained corresponding to each value of pitch shown in the table. These values are subtracted from the corresponding numbers giving the hearing loss. The number in the table corresponding to the resulting value of $H-A$ can now be found. These values of ΔM are all for the case when the speaker is at a distance of one meter from the microphone of the hearing aid. For distances of $\frac{1}{2}$, $\frac{1}{4}$, $\frac{1}{8}$, $\frac{1}{30}$ or $\frac{1}{100}$ of this distance add 6, 12, 18, 30 or 40 decibels to the amplification factor and then proceed as before. Also for distances greater than one meter, namely, for 2, 4, 8, 30 or 100 times this distance, subtract 6, 12, 18, 30 or 40 decibels from the amplification factor A .

To illustrate this method of calculation, let us calculate M for patient No. 8, whose audiogram is shown in Fig. 3, when he is

TABLE I.
VALUES OF ΔM .

HEARING LOSS MINUS AMPLIFICATION H-A															
Frequency Pitch	-5	0	5	10	15	20	25	30	35	40	45	50	55	60	
250	-2.0 0.008	0.008	0.007	0.006	0.005	0.004	0.003	0.002	0.001	0	0	0	0	0	
350	-1.5 0.044	0.044	0.041	0.038	0.034	0.029	0.023	0.016	0.010	0.005	0	0	0	0	
500	-1.0 0.076	0.075	0.072	0.068	0.064	0.056	0.048	0.038	0.027	0.019	0.010	0	0	0	
700	-0.5 0.091	0.091	0.089	0.086	0.082	0.075	0.066	0.056	0.045	0.034	0.024	0.011	0.008	0	
1000	0.0 0.103	0.103	0.103	0.102	0.099	0.094	0.086	0.077	0.065	0.053	0.040	0.026	0.014	0.001	
1400	0.5 0.116	0.116	0.116	0.116	0.115	0.111	0.106	0.097	0.086	0.071	0.055	0.040	0.025	0.013	
2000	1.0 0.169	0.169	0.169	0.169	0.167	0.162	0.153	0.139	0.116	0.095	0.071	0.051	0.029	0.011	
2800	1.5 0.151	0.151	0.151	0.150	0.147	0.140	0.128	0.107	0.087	0.064	0.041	0.023	0.007	0	
4000	2.0 0.131	0.131	0.130	0.126	0.118	0.105	0.086	0.066	0.047	0.028	0.001	0	0	0	
5600	2.5 0.106	0.104	0.099	0.083	0.069	0.054	0.040	0.026	0.012	0	0	0	0	0	

M = \sum Δ M

$$M = \Sigma \Delta M$$

using the hearing aid labeled B in Fig. 5 for the condition when the speaker is one meter from the microphone. The calculation is shown in the table below.

TABLE II.
PATIENT NO. 8. HEARING AID B.

Pitch	Amplification A Curve B (Fig. 5)	Hearing Loss Audiogram 8	H-A	ΔM With Aid	ΔM Without Aid
-2.0	30	40	10	0.006	0.000
-1.5	29	44	15	0.034	0.001
-1.0	28	48	20	0.056	0.004
-0.5	30	60	30	0.056	0.000
0.0	40	70	30	0.077	0.000
0.5	30	70	40	0.071	0.000
1.0	23	70	47	0.060	0.000
1.5	30	70	40	0.064	0.000
2.0	-5	70	75	0.000	0.000
2.5	—	—	—	—	—

$$M \text{ (with aid)} = \Sigma \Delta M = .424$$

$$M \text{ (without aid)} = \Sigma \Delta M = .005$$

$$\text{Distance} = 1 \text{ meter}$$

The values of A and H for each of the ten values of P are shown in columns II and III. The values of ΔM obtained from the table corresponding to each pair of values of P and H-A are shown in the next to last column. Adding the numbers in this column together gives us the values of M, namely .42. The numbers in the last column correspond to A = 0, that is, to the condition when no aid is used. This index figure is related to the per cent of test sentences which the patient would interpret correctly *after having sufficient practice* with the set. This relationship is shown by the curve in Fig. 6.

It must be emphasized that the phrase "after having sufficient practice" was underlined in the above statement. It will be recognized that any consideration of this kind must be based upon averages and in this instance upon averages obtained with persons of normal hearing. Our experience with a very limited number of hard of hearing persons seems to indicate that such a curve shows the expected attainment when using such a hearing aid. For patient No. 8, then, the above calculation shows that without an aid the patient will not be able to understand any speech but will obtain satisfactory aid with set B. Also these calculations assume that room reflections do not change the result. At the

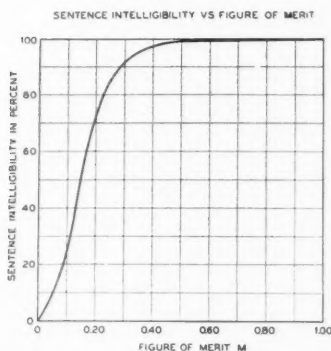


Fig. 6.

larger distances the reverberant effect of the room may produce an effective value of M which may be as low as one-half that calculated. At distance shorter than one meter this room effect is small. As a general working basis, it may be stated, that conditions and sets giving M values greater than 0.4 will give excellent results, those having values between 0.4 and 0.2 will be satisfactory while those below 0.2 will be unsatisfactory. For example, for patient No. 11, having the audiogram shown by the solid curve in Fig. 4 when using either set A, set B, set C, or using no aid, the value of M for various distances is given in Fig. 7 by the solid curves.

These calculations are based upon the assumption that the speaker will use a normal conversational type of voice. If the intensity of the voice is made greater than this, it will have the same effect as increasing the amplification. One can only raise his voice about 15 db above the normal and still maintain good pronunciation. The dotted curve in Fig. 7 is for the case when the speaker uses about as loud a voice as he can, namely, 15 db above the normal.

It will be seen that with the unaided voice, the patient No. 11 will only be able to hear and understand at a distance less than $1/32$ meter—that is, 3 cm., and then only when a shouting voice is used. With set A he should be able to carry on a conversation with a microphone at from $1/4$ to 1 meter distance. Tests with this patient and hearing aid indicated that this was possible.

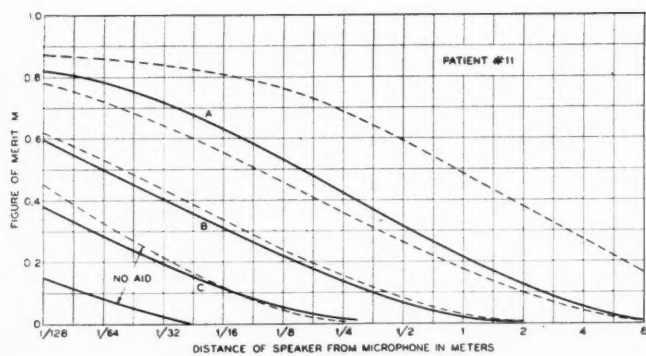


Fig. 7.

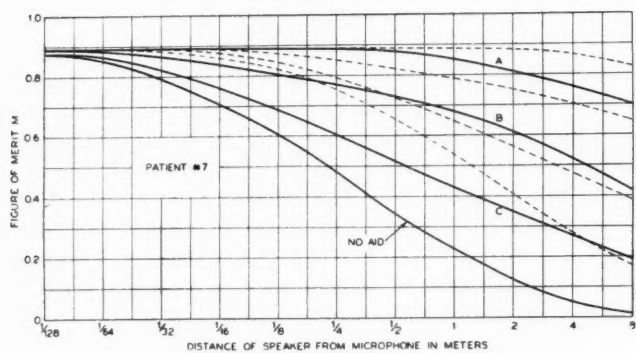


Fig. 8.

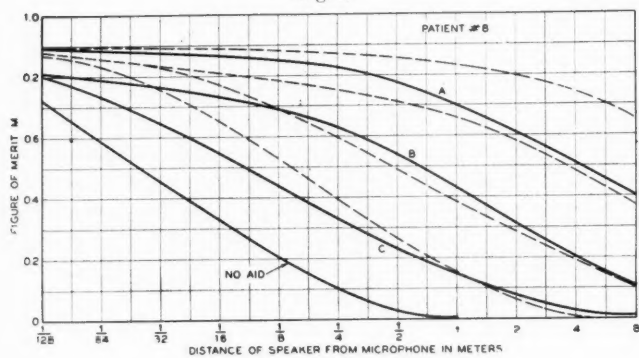


Fig. 9.

In Fig. 8 the values of M for patient No. 7, using sets A, B and C are given. It will be seen that he will be able to converse with some difficulty for distances from 1.5 to $\frac{1}{2}$ meters and with no difficulty for distances less than $\frac{1}{2}$ meter without any help from a hearing aid. Either of the three aids A, B or C will give considerable help to this patient for any distance usually encountered in a small room. Set A should give considerable help in a large auditorium provided the room acoustics are not too poor. Similar curves for patient No. 8 are given in Fig. 9.

These calculations are based upon the assumption that the amplification curves given in Fig. 5 hold for all distances. This is true for some types of sets, but far from true for others. So to predict the usefulness of a set the operating distance of the set must be known as well as the amplification curve. For example, the set B, discussed above, becomes almost inoperative for large amounts of speech power corresponding to normal talking at distances closer than $\frac{1}{8}$ meter and also for small amounts of speech power corresponding to normal talking at distances greater than about four meters.

Also, in a more accurate theory, account must be taken of the fact that for all ears the speech becomes distorted more and more by the hearing mechanism as the intensity of the sound at the ear increases. This distortion differs for different types of deafness. For the general purposes outlined in this paper, this may be neglected.

It is evident that in such a short paper only some very general considerations can be given concerning this problem. It was my purpose to point out some of these general principles to guide one in making recommendations. To apply even these general considerations would be necessary to have accessible such amplification curves as shown in Fig. 5 for all the sets which are commercially available.

In conclusion, may I suggest the possibility of appointing a committee from this society to obtain such curves and make them available to members. If this is done, I hope that some of you will be sufficiently interested to try the procedure outlined above so that it may be checked against actual clinical experience.

BELL TELEPHONE LABORATORIES.

LVIII.

PHYSICAL DATA AND PHYSIOLOGY OF EXCITATION
OF THE AUDITORY NERVE.*

R. L. WEGEL,

NEW YORK.

INTRODUCTION.

The understanding of the mechanism of hearing is at present inadequate, from the standpoint of general utility. In part, this state of affairs is due to the lack of co-ordination of available information concerning the physiology of excitation of tissue and the numerous functional physical data. Physical data, such as absolute acuity, pitch and intensity discrimination, masking, etc., are easily obtained in quantity and with precision, but cannot be properly utilized without correlation with physiologic data on tissue excitation and propagation. The measurement of such a property, for example, as absolute acuity or ear sensitivity should be regarded as an experiment in physiology and not as an experiment in physics, physical apparatus being merely a tool. It is the purpose of this paper to summarize the results of a semi-quantitative preliminary inquiry into the nature of a correlation between "physical" data on hearing and the excitation properties of tissue.

The ear may be regarded, for convenience in this paper, as having three functionally distinct parts: the purely mechanical apparatus, the nervous tissue consisting of the auditory nerve tract with all its ramifications into the cochlea, and the receptors, presumably the hair cells, which act as transducers or transformers of physical stimulus of motion into nerve impulses. These are roughly analogous in the eye, respectively, to the optical system, the optic nerve, including the ganglion cells and their ramifications in the retina, and the complex mechanism of the retina which acts as a transducer of light stimulus into nerve impulses.

The problem of hearing is no exception to the rule, in the study of sense organs, that the receptor mechanism is the most difficult

*Presented before the American Otological Society, Atlantic City, May 17-19, 1932.

element in this conducting chain to approach experimentally. The properties of these intermediate elements must be determined by indirect inference from experiments performed directly on the complete organ and from measurements made directly on the nervous tissue, all in the normal living condition so far as possible. Circumstances suggest that the hair cells function in response to mechanical motion to produce electrical excitation of the nerve terminals.

This paper is limited to the consideration of certain features of physiologic data on direct excitation of tissue and to certain physical data taken on the complete organ. The outline is as follows:

DIRECT EXCITATION OF TISSUE.

1. Mechanism of excitation.
2. Single impulse excitation—diffusion or physical phase.
3. Formulæ for calculating excitation in the diffusion phase.
4. Multiple impulse excitation—extra diffusion or metabolic phase.

OVER-ALL MEASUREMENTS ON THE HEARING ORGAN.

1. Absolute acuity for pure tones.

It is planned to go into the matter of continued iterated excitation of tissue, intensity discrimination, accommodation and binaural phenomena at a later time.

DIRECT EXCITATION OF TISSUE.

This outline is a collection of those available data and theories of physiology which appear at the present time to be most pertinent to the problem of excitation of the auditory nerve through the medium of sound stimulus and the intermediate step through the auditory receptors. It is limited to such information as is concerned with tissue excitation and recovery and is presented in a form to provide concepts more readily applicable to the problem in hand. The physiological data are as yet not adequate for theoretical treatment in the sense of the methods of modern physics.

1. *Mechanism of Excitation.*—The experimental physiologic data to be described is obtained largely from nerves and muscles of lower animals and the question will naturally arise concerning the bearing of this on the functional properties, specifically of the cochlear nerve. There is good justification for the assump-

tion that in so far as the gross features of excitation and propagation of action are concerned these tissues differ only in sensitivity and times of reaction.¹

The similarities of tissues in excitation and propagation are demonstrated in many ways which cannot be described here. At the expense of overdue emphasis, an interesting correlation, due to R. S. Lillie, is described.

In 1914,² R. S. Lillie collected a considerable number of observations published by various authors which tend to indicate a definite relationship between the duration of the rising phase of action current and the velocity of propagation of action in different tissues. Fig. 1 is a logarithmic plot of the values of propagation velocity against the duration of the rising phase taken from these data. The points plotted represent definite values given. The rectangles represent estimated ranges as given. Data for which one value for τ is given with a range for v are plotted as heavy vertical lines. These are enclosed by dotted circles to suggest a probable total range. These circles may be accepted or not, as desired, as proper representation of the data.

It may be seen from the figure that the data covers a range of velocities of from .1 to 10,000 cm/sec., a ratio of 1:100,000, and a range of rising phase duration of from .0005 to 2 sec., a ratio of 1:4,000. The quickest represents a high speed nerve and the slowest a very low speed muscle, both of which may be taken

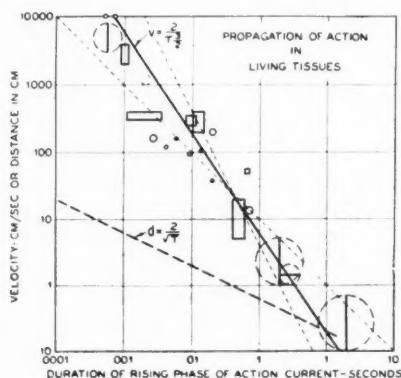


Fig. 1.

from the same animal. The intermediate values represent nerves and muscles taken from various animals. A few narcotized tissues are also included. A line is drawn through the points, which is suggested as the best single representation of the data. It will be seen that a few observations are not as accurately on the curve as might be desired, but it should be borne in mind that the sources are unrelated; also that, according to recent work of Erlanger, Gasser and Bishop, any tissue is in general a complex of elements of various speeds, one of which presumably predominates in such measurements, and that there is no uniformity in the kinds of electrodes used. This curve graphically illustrates Lillie's contention that there is something in common in action propagation in all tissues.

The slope of the curve is 3:2, and is represented by the equation

$$v = \frac{c}{\tau^{3/2}} \text{ or } v\tau = \frac{c}{\sqrt{\tau}} \quad (1)$$

In these expressions c is a constant having a value of about 1/5.

In order to test the degree of dependability of the $3/2$ power law as a representation of the data as given, two additional lines are drawn dotted having slopes of 1:1 and 2:1, having equations respectively

$$v = \frac{c}{\tau} \text{ and } v = \frac{c}{\tau^2} \quad (2)$$

These lines illustrate, if a single law represents the data, that it must be between the inverse and the inverse square—that is, the inverse $3/2$ power law or near it. In order to determine the $v - \tau$ relation with precision a special investigation seems to be called for.

It is of interest to note in this connection that Lapicque³ has preliminary evidence to show that the velocity of propagation of action is 1 cm. per chronaxie approximately, and suggests that the duration of the rising phase is a constant number (three) of chronaxie. This would imply an inverse proportionality, as suggested by Lillie, between velocity and rising phase, which is apparently not in accord with these data.

The product $v\tau$ of equation (1) is the linear distance, measured on the tissue surface along the line of propagation, which is

occupied at any instant by the rising phase of action current. This is also plotted in Fig. 1 and marked *d*. It varies as the reciprocal of the square root of duration of the rising phase. The range is from about 10 cm. for the most active tissue to about 1 or 2 mm. for the slowest, a ratio of the order 1:100. It is to be noted that if the law $v = C/\tau$ were correct, the range of action *d*, or $v\tau$, would have a constant value of about 1 cm. for all tissues. From Eq. (1) it may be seen that the velocity of propagation is proportional to the cube of this distance, or that the distance is proportional to the cube root of the velocity.

Any single law expressing the relation between two functional aspects, such as *v* and τ , in an assortment of tissues gives a certain amount of information concerning the rules used by nature in making them physically and functionally different. There are as many such rules as there are essential physical components in the mechanism. In order to utilize an assortment law, such as (1) quantitatively it is necessary first to know the special mechanism of propagation of action. The laws given in Eqs. (1) or (2) may apply equally well to any kind of propagation, such, for example, as of flexural waves in a properly chosen assortment of bars or to gravitational waves in liquids. These data contribute to the conclusion that all tissues are fundamentally alike as concerns the mechanism of excitation and propagation.

The surface of the cell is considered to be the seat of sensitivity to external stimulus. This is, of course, only an approximation, but useful as a tentative working hypothesis. Physically, a living cell consists of an aggregate of individually complex structures, each of which has a primary function which is in general different from that of any other. Each element performs its functions, as a component of the cell by virtue of the properties of its surface, the interface between its internal material and that external to itself. Excitability or irritability is localized principally at the gross surface of the cell and the maintenance of the proper surface condition for this function is dependent on metabolic activity within the cell and physical external conditions.

The surface of a resting cell is considered to constitute a barrier between a surplus accumulation or "atmosphere" of negatively charged ions on the inside and a similar surplus of positively

charged ions on the outside. This makes it a "Helmholtz double layer." The properties of tissue which, by one means or another, maintain the separation of the charges at this surface against their attractive forces, make it resemble in some respects a semipermeable "membrane," which designation is frequently used without the implication that a physical skin or wall, in the ordinary sense, necessarily exists. There are certain ingredients in the protoplasm of the cell which easily combine with oxygen furnished normally from the outside. When such a combination takes place at the cell surface a partially protective layer, of composition at present unknown, is formed bringing the cell into a condition analogous, as described by Lillie, to the passivity of certain metals in oxidizing solutions. In this passive or partially passive condition the cell is at "rest" or in dynamic (not "thermodynamic") equilibrium with its surroundings. A removal or attenuation locally of this protective film wholly or partially restores activity. A small attenuation of the film results in an acceleration of chemical action, more or less locally, to restore it to the passive state. When the film is suddenly removed, leaving the surface clean or active, the potential across it disappears, leaving it relatively permeable. In this condition the excited or active surface acts as a short circuit on the undisturbed surface next to it, resulting in a discharge, like that of a battery, of the undisturbed portion through the excited part. As a consequence, the region which has been so discharged becomes partially active or excited and the region through which the discharge has occurred assumes a degree of passivity. This process is repeated from one excited area to the next and constitutes a propagated "wave" of action. This mechanism may be likened to that of a series of batteries, having certain hypothetical properties, connected in parallel with resistances between them so adjusted that if the potential of the first is reduced by a discharge the second discharges through it, permitting the third to discharge through the second, and so on in succession.

The condition as to degree of passivity of the cell surface, which may also be thought of roughly as the reciprocal of its "permeability," is subject to considerable variations induced by external influences. Mechanical contact of an inert object cleans or activates it and may cause excitation. Distortion of the surface atten-

uates the protective film at certain places and may result in excitation. Increase in temperature tends to dissipate the passive layer, making it more sensitive and, if effected quickly, may cause excitation. Immersion in certain electrolytes causes either an increase or a decrease in passivity, according to the nature of the electrolyte.⁴ If an electrical current is passed through the tissue in such a way as to produce a potential across a cell surface at some point, an excitation or activation will be produced at that point if the potential is high enough, suddenly enough applied, and in the proper direction to neutralize the charge there existing.

The observed properties of tissues in response to stimulus may be interpreted to describe certain features of the membrane "batteries" which cannot be very well determined in any other way. It is necessary to know these properties in order to discover the nature of the functional properties of the receptor hair cells in response to sound stimulus. A discussion of these properties is best deferred until after an examination of the typical data.

2. *Single Impulse Excitation.*—Among the first quantitative experiments to be done was the measurement of effectiveness in excitation of single electrical impulses of various intensities, durations and shapes applied directly to the tissue. The largest single sources of present information are Lapique and Keith Lucas.⁵ Many important investigations of more discriminatory nature have been in progress during recent years.

In applying electrical stimuli to gross tissue two electrodes of special design (nonpolarizable) are applied at some distance apart and current from a convenient source passed through the tissue. Of these two electrodes, the cathode which is a source of negatively charged ions produces a potential across any cell surface in the current path, which tends to neutralize the charge existing on it and hence to activate or excite it. The anode has the opposite effect. This potential drop of the stimulating current across the cell wall is due to that fraction of the total current which passes through it, traveling longitudinally along the cell and out through the wall near the other electrode.

Excitation originates at the cathode on the "make" of the current. It is also observed that when the applied current is strong enough a second excitation occurs on breaking the stimulating

current, but this time originating at the anode. It may be supposed that on applying the current, the tissue in the region of the anode is temporarily abnormally passivated, as the external potential on the membrane is in such a direction as to require it. A rough qualitative picture may be had by supposing that the potential on the resting membrane is $+e$ volts and that the stimulating circuit superposes a neutralizing potential of $-e$ volts near the cathode resulting in time in excitation there. At the same time an additional potential of $+e$ volts is superposed near the anode, making a total there of $+2e$ volts and leaving the tissue at that point in a higher state of passivity than before. Now after a certain interval of time this excess potential produces an inhibiting effect on the electrochemical process within the cell, which is the normal passivating agent and eliminates it to a considerable degree. The result is that after a time the tissue is here left with a passivating potential of about $+e$ volts and this is furnished externally by the applied anode. When the circuit is broken this also disappears, leaving the surface in an active condition, and so excitation occurs at the anode on "break." Previous to this time an impulse having originated at the cathode, the potential $-e$ is maintained there by the stimulating circuit, and the tissue soon chemically builds up a potential $+2e$ which, superposed on $-e$ of the external circuit, leaves the net potential there $+e$, in which condition the tissue is near the normal passive state. On breaking the circuit the $-e$ due to the cathode disappears, leaving $+2e$ due to the cell metabolism which results temporarily in a superpassive state. This in time readjusts itself automatically to $+e$ volts and thus no excitation originates at the cathode on breaking the circuit. This conception throws the burden of a considerable part of the properties of tissue on the electrochemical action which, in the resting state, proceeds with a certain velocity and under conditions of external stimulation has its rate changed or catalyzed in one direction or the other.

In an experimental study of excitation of tissues, cathodic stimulus is almost always used, so provision must be made to eliminate the effects of the anode. When it is desired to study the action of a nerve by observing its effect on an attached muscle, it is common practice to kill a short length of the free end of the

nerve and apply the anode there while the cathode is brought in contact with the live nerve trunk. Experimenters assume that the perturbing effect of the junction of living and dead tissue is to be disregarded.

Fig. 2 is a logarithmic plot of excitation data taken by Lapicque with special care for accuracy on two different specimens of a frog's sciatic—gastrocnemius—preparation.⁶ The nerve is stimulated with a "rectangular" electrical impulse (sometimes unfortunately called constant current). This is done by abruptly applying current of a measured intensity, holding it constant for a time which is just necessary to excite and measuring this time interval. Referring first to experiment 2 of Fig. 2, it will be seen that

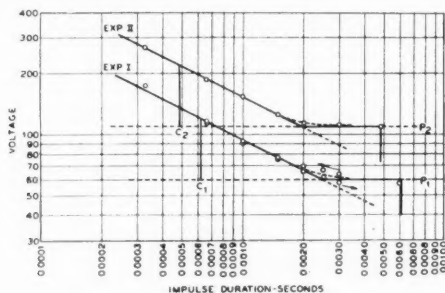


Fig. 2.

excitation occurs if 270 volts are applied to the stimulating circuit for an interval of .00033 seconds, 187 volts for .00066 seconds, 155 volts for .001 seconds, and so on. A voltage less than about 110 (111.5 is the figure given) fails to excite, no matter how long applied. This minimum voltage is effective in about .004 to .005 seconds and is represented in the diagram by the end of the horizontal line. This minimum value of voltage has been given the convenient name "rheobase" by Lapicque, and is designated by q in this figure, with the subscript 2 to refer it to experiment 2. The actual value of this voltage is of no significance, as these figures do not represent current passing through the cell membranes, nor even the available voltage at the gross tissue surface. Thus although the rheobasic stimulus is 111.5 volts as applied to the exciting circuit, the corresponding voltage across

a favorably situated cell membrane is probably of the order of magnitude of 100 millivolts. Although it was not to be expected, there is fairly good experimental evidence to show that to a close approximation, the current density through any cell wall in the path of the current is proportional to this voltage.

By examining the curve for experiment 2 it will be seen that in proceeding from shorter to longer intervals the points lie closely on the sloping straight line up to about .0015 seconds. From this point they follow a curve which quickly approaches the horizontal or rheobase line. The measurements in these two cases were made on the tissue by testing first at an interval duration of current of .003 seconds, then gradually decreasing the interval to .00033, and returning, making the final reading on the rheobase. The points plotted for experiment 2 are the average of two readings taken for each interval. In experiment 1 the actual readings are plotted. For the longer intervals, where the time between tests is large, it may be seen that the voltage values were about 10 per cent higher at the beginning of the experiment than they were on returning down the curve, but for the short intervals the readings are practically the same. Lapicque estimates that his values are reproducible to within about 5 per cent, so the indications are that the tissue has drifted to a more sensitive condition during the progress of the experiment due either to the excitation or to some other cause. The average values, as calculated by Lapicque and plotted in curve 2, are said to be a good representation of the tissue in a constant state.

The actual value of the time units are of considerably more significance than those of voltage. The time corresponding to the intersection of the sloping and horizontal parts of this excitation curve is .00195 seconds for experiment 2 and about .0025 seconds for experiment 1. It has been found that, provided the same type of electrodes are always used, and the tissue tested under constant experimental conditions of age, temperature, etc., the time corresponding to this intersection which locates the curve on the time scale is relatively constant and characteristic for any given tissue. In order to express this characteristic time constant for a given tissue Lapicque proposed the definition of the interval required to excite when the voltage was double rheobasic strength

and named it the chronaxie. This is designated c_2 for experiment 2 and has a value of .00049 seconds, which it will be seen is one-fourth the value corresponding to the intersection of the sloping and horizontal portions of the curve. This definition of chronaxie was chosen because of the ease of its direct measurement without plotting a curve. The effective duration of voltage at rheobasic strength has been found to be about 8 to 10 chronaxie.

There is one characteristic of this curve which is of considerable theoretical importance and that is its shape. As long as rectangular impulse stimulus is used and electrodes employed which are large in extent compared with the ultimate excitable cell and not placed in too close contact with it and, further, as long as the stimulated tissue is reasonably homogeneous in a sense that all elements (cells) within it have about the same reaction time, the shape remains substantially unchanged from tissue to tissue. Lapicque has measured such curves for tissues having chronaxies varying from a few ten-thousandths of a second to several seconds, and always finds substantially the same shape. He has even found such curves to represent the excitation of protozoa and of plants. The shape evidently has to do with tissue excitation in general. The indication of such circumstances was pointed out and treated theoretically by Nernst.⁷ Since a knowledge of this quantitative phenomena is necessary in inferring the effect of the hair cells on the terminals of the auditory nerve, it is advisable to go into the matter briefly here.

By examining either of the curves of Fig. 2, it will be seen that the inclined straight line has a slope 1:2, so that when the time is divided by 4 the stimulus is doubled. It might be noted here, parenthetically, that many measurements in some cases show slight but systematic deviations from this 1:2 slope. The portion of the data corresponding to intervals of 3 or 4 chronaxie or less are, therefore, represented by the law

$$E = \frac{K}{\sqrt{\tau}} \text{ or } E\sqrt{\tau} = K \quad (3)$$

Here E represents the voltage, τ the duration of the interval to correspond, and K a constant. If the reacting effect of locally changing polarization be disregarded, the stimulating current

density anywhere in the tissue is proportional to this voltage which produces it, so it is concluded that for any interval shorter than three or four chronaxie the stimulating current density passing through any cell surface requires a time to excite which is inversely proportional to the square of the current, or that the intensity of current density required to excite is inversely proportional to the square root of the time interval.

In developing a theory of excitation Nernst limited himself to the application of his theory, previously successful in electrochemistry, of diffusion of electrolytes and specified that this applied only for these shorter time intervals where the square root law holds. Unfortunately for our present purpose, he made no attempt on the phenomenon for more protracted excitation time but explained, without giving details, that for these longer intervals an "accommodation" of tissue occurred. Nernst's theory is that of migration of ions, known to exist in the tissue, under the influence of an externally applied electric field and subject to the laws of diffusion in dilute solutions. Under these circumstances he calculated the current and time required for ions to accumulate at a cell surface impermeable to one ion and permeable to the other in sufficient quantity to neutralize a charge already existing there and found the square root law. In his description this matter of neutralization is not explicitly stated. In order to work out a mathematical theory of such a system the geometrical boundaries of various parts of the structure must be specified. He did not give an elementary description of this phase of the problem but, concerning nerve excitation, the picture he had in mind was probably as follows:

An electrode is applied to a moist nerve trunk, more or less cylindrical in section. Current enters this trunk directed generally inward at the position of the electrode. It then swings about along the axial direction toward the other electrode. The only possible carriers of current are ions. The current, in flowing down the trunk, tends to spread so as to occupy its entire area and hence to penetrate the axon surfaces and pass internally along them. Such part of this radial component of charge as is thus carried to a cell membrane by the ions in an effort to pass inside the cell constitutes the excitant for that cell, and it is presumed

that when they are accumulated there in quantity to sufficiently neutralize excitation occurs.

The difficulties of rigorous mathematical analysis of this structure under the actual conditions of stimulation are so great that it is hardly worth attempting it, especially in view of the present status of experimental knowledge, so recourse is had to more practical idealization of the structure. For this purpose Nernst considered that the time intervals in question were so short that the effects in depolarizing the cell surface by diffusion were effective only at distances from this surface which are small compared with the diameter of the cell. It is known from the properties of the diffusion process, as represented by the properties of the solution of the diffusion equation, that for short enough time intervals the theoretical assumption that the cell surface is plane and infinite in extent and that the protoplasm is infinitely thick gives the same results as a rigorous analysis. When Nernst found that his theoretical formula agreed with the form of the curve up to time intervals of about 3 chronaxie he considered his assumption to have been justified up to that point.

Some time later, A. V. Hill⁸ made the further step in the direction of a rigorous solution. He also gave a "one-dimensional" treatment of the problem and considered only that part of the current which traversed the cell. His assumed structure idealized the axons as infinite flat sheets of thickness such that, for time intervals up to perhaps 5 or 10 chronaxie, the ion had time to traverse an appreciable part of the cell thickness. This gave a theoretical formula, intuitively satisfactory, agreeing with the square pulse data as far as it went. It implied the assumption, however, that the observed deviation from the square root law at about 3 or 4 chronaxie was due to this effect of thickness and not to a change in the metabolic processes which Nernst referred to as accommodation. The general magnitudes of diffusion coefficients are such that the order of magnitude of expected depth of penetration is not safely small enough to eliminate from consideration this thickness effect as a deviating factor on the excitation curve for long time intervals.

To summarize, although the data do not always follow the square root law with precision, it is generally agreed that for

intervals shorter than about 3 chronaxie the Nernst diffusion formulae represent the best practical working approximation available. For long intervals, of minimum length unspecified but longer than 3 chronaxie, changes in metabolic rates influence the effectiveness of a stimulus in excitation. For intervals, of lengths unspecified but longer than 3 chronaxie, the Hill thickness effect plays a part. At what time specifically the last two effects become appreciable and to what relative extent, is not very clear. For extremely long intervals, say certainly from ten chronaxie up, some sort of accommodation process is a controlling factor.

3. *Formulae for Calculating Excitation in the Diffusion Phase.*

—Since the value in the hearing problem, of excitation data on tissues, will depend largely on the ability to calculate the effectiveness of stimuli of forms other than those so far measured, it is necessary to reduce the information to simple formulae as far as possible. Complicated formulae are of little use. The Nernst conception yields formulae which in general are in best accord with the known properties of electrolytic conduction and with the physiologic data within the limits of the diffusion or physical phase of excitation.

Excitation data are frequently taken with impulses which are not of the rectangular or "constant" current form. In particular, the relaxation or exponential pulse is much used because of its simplicity of control. This is obtained by discharging a condenser through a resistance and leading a part of the resulting current through a suitable circuit to the tissue. The time intervals are varied by changing the value of the condenser in such a way as to change the effective duration of the discharge in a predetermined way. Although this stimulus never exactly vanishes, it decreases in a time, measured by the "time" constant of the circuit, to a negligible value. When a single impulse is used, excitation occurs at or about the time during the discharge when the concentration of ions at the cell surface reaches the excitation value. Alternating currents have also been frequently used.

Within the diffusion phase the simple formulae, applying approximately to concentration at a boundary surface of ideal differential permeability have been developed to apply to stimuli of any form by Dr. S. R. Milner.⁹ The formulation by Milner may be

modified slightly for convenience. It holds good at such an ideal boundary of any diffusion medium for which conditions are such that the continuity is expressed by the simple diffusion equation. This approximation has been assumed in all excitation theory.

When a current i in effect flows into the boundary of a diffusion medium for a time t the concentration of ions at the boundary is found to be proportional to a quantity y given by

$$(A) \quad y = i \sqrt{t} \quad (4)$$

This is in accord with the rectangular pulse data of Fig. 2 and equation (3).

When a current, say i_1 , flows for a time t_1 , then suddenly changes to another value i_2 , which continues unvaried up to a time t_2 , then becomes abruptly i_3 , up to a time t_3 , and so on, the degree of concentration at some later time t is given by the formula

$$y = i_1 \left\{ \sqrt{t} - \sqrt{t-t_1} \right\} + i_2 \left\{ \sqrt{t-t_1} - \sqrt{t-t_2} \right\} \\ + i_3 \left\{ \sqrt{t-t_2} - \sqrt{t-t_3} \right\} \\ \text{etc.} \quad (5)$$

Each term represents the contribution at the time t to concentration made by each step, previously made, in the current. An equivalent form, which is usually simpler for calculation, is

$$y = i_1 \sqrt{t} + (i_2 - i_1) \sqrt{t-t_1} + (i_3 - i_2) \sqrt{t-t_2}, \text{ etc.} \quad (6)$$

When the current changes smoothly an approximate value of y may be obtained by the use of (5) or (6) by dividing the time t into a number of intervals and writing for i_1, i_2, i_3 , etc., the average value of current during each interval. It frequently happens, however, that the more exact integral form is more convenient. This is readily obtained from (5).

$$y = \int_0^t \frac{i(\lambda) d\lambda}{2\sqrt{t-\lambda}} \quad (7)$$

In this form λ is the time variable of integration corresponding with t_1, t_2, t_3 in the above series and $i(\lambda)$ is the current expressed

as a function of the time λ . If i is constant up to a time t at which it is desired to evaluate y , then $i(\lambda)$ is constant through the range of integration and the expression (4) is obtained.

It is interesting historically to note that an equivalent form of (7) obtained from the series (6) is

$$y = \int_0^t \frac{di(\lambda)}{\sqrt{t-\lambda}} d\lambda \quad (8)$$

This is a specific form of the old empirical law of DuBois Reymond and is useful in types of impulse where formula (7) is less convenient or inapplicable. This form cannot be used for a rectangular impulse nor for any impulse in which the stimulating current has discontinuities. The reason for this limitation will be evident by examining the process by means of which it is derived from the equivalent series (6).

In case of a condenser discharge (this was worked out by Euker working with Nernst in 1910-12), the stimulating current is given by

$$i = i_0 e^{-\Delta t} \quad (9)$$

where i_0 is the initial value of current and Δ the relaxation constant of the circuit ($\Delta = 1/RC$ where C is the capacity and R the resistance of the discharge circuit). To calculate y , the degree of accumulation of excitant, replace t by λ in (9) and substitute in (7).

$$y = \int_0^t \frac{i_0 e^{-\Delta \lambda}}{2\sqrt{t-\lambda}} d\lambda \quad (10)$$

If for simplicity time be measured in units of $1/\Delta$ and this integral be transformed by substitution $x = \sqrt{t-\lambda}$

$$y = i_0 e^{-t} \int_0^{\sqrt{t}} \frac{\sqrt{t}}{e^{x^2}} dx \quad (11)$$

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If for simplicity time be measured in units of $1/\Delta$ and this integral be transformed by substitution $x = \sqrt{t-\lambda}$

$$y = i_0 e^{-t} \int_0^{\sqrt{t}} \frac{\sqrt{t}}{e^{x^2}} dx \quad (11)$$

This is a definite integral, the values of which have been tabulated.¹⁰ The values of y , the accumulation of excitant, are plotted as a function of time measured in units of $1/\Delta$ in curve y -B, Fig. 3. The curve i -B is a plot of the stimulating current as it varies with time. It will be seen that the stimulating current decreases continuously with time but that the excitation rapidly accumulates at first, reaching a maximum value at a time $.853/\Delta$ and then decreases slowly. In contrast with this, the accumulation y due to the rectangular impulse, marked y -A, shows a rapid initial rise similar to that of the exponential pulse but continues indefinitely to accumulate as the pulse is prolonged. The excitant accumulates in response to a rectangular impulse to a value equal to that of the maximum for the exponential pulse at a time

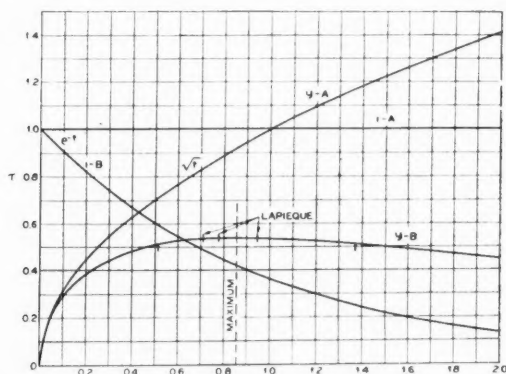


Fig. 3.

$.292/\Delta$. The ratio $.292/.853$ is $.344$. Thus, if it is desired to know the duration of a rectangular exciting pulse having the same current or voltage as the initial value of the exponential pulse, it is found by measuring the time constant of the exciting circuit and multiplying this by $.344$.

In exciting with the condenser discharge it is customary to set the time constant and then to vary the voltage until excitation is just produced. When this is done with pulses short enough to be included within the simple diffusion phase, excitation must occur at or about the time of maximum accumulation—that is,

.853/ Δ . It will be seen that the curve shows so broad a maximum that a very small error in determining the voltage results in a large error in determining the effective time, or "*temps utile*," of the condenser discharge. This difficulty of precision does not exist with the square pulse. Lapique has measured this ratio of effective times and obtained a range of values .31 to .41. He adopts as an empirical average .37. The points corresponding to Lapique's value for effective times .31, .37, .41, as well as .34, the position of the maximum, are indicated on the curve and it will be seen that over the entire range the required accumulation varies almost imperceptibly. Lapique has also attempted a direct measurement of the useful part or effective time of the condenser discharge. His values, which should theoretically be .853/ Δ , vary over a very large range. He has determined by experience that the accuracy of determining a voltage usually runs about 5 per cent when the experiment is carefully done. Assuming this figure for accuracy, his measured values should be expected to fall between time intervals where the excitant is 95 per cent of the maximum value—that is, .541, or from $t = .51/\Delta$ to $1.37/\Delta$. Two series of experimental values are given: .75, .52, .60, .86 (April 26th); .71, .68, .71, .86, 1.16 (April 28th), all of which, in spite of the wide experimental range, are within the expected time limits.

On the whole, the data seem to be as well in accord with the theory as might be expected in view of the various factors necessarily neglected for convenience in formulating it.

Some measurements have been made¹¹ with impulses having an initial linear increase but no measurement of the useful or effective time is available. For the linearly increasing current put $i = m\lambda$ in equation (7) or (8), and the resultant excitation is predicted to accumulate according to

$$(C) \quad y = \frac{2m}{3} t^{3/2} \quad (12)$$

Comparing this with (4) it will be seen that within the diffusion phase, to excite in the same time, the linearly increasing current must rise to a value 50 per cent greater than the rectangular pulse. For the increasing current to excite when it reaches the

same value as that of the rectangular pulse takes $9/4$ or a little over double the time.

Measurements have also been made with ascending exponential stimulus $i = i_0 (1 - e^{-\lambda t})$, which when substituted in (7) or (8) gives

$$(D) \quad y = i_0 \sqrt{t} - i_0 e^{-t} \int_0^{\sqrt{t}} e^{x^2} dx \quad (13)$$

This is merely the difference between (A) and (B). Experiments with this impulse are, in appearance at least, not as well in accord with the data as might be desired, but they are not always clear as to the duration as compared with the chronaxie.

It would seem that the ideal pulse for experimental purposes would be one in which the excitant y increases linearly with the time. Such an impulse may be obtained by making the stimulating current proportional to the square root of time. Thus, if $i = a\sqrt{t}$

$$(E) \quad y = \frac{\pi a}{4} t \quad (14)$$

This could probably be obtained with an artificial "diffusion" network consisting of a series of resistance shunt capacity elements, across which the voltage was tapped off while being charged through a high resistance. The theory of design of such a circuit should not be difficult.

For use in calculation the following table giving values of y for various types of pulses as a function of time is given. The letters refer to the types of impulse. The time units in each case are those given in the formulæ above.

4. *Extra-Diffusion or Metabolic Phase: Multiple Impulse Excitation.*—When a succession of short impulses of stimulus are so spaced that the total duration of the train is less than 3 chronaxie, the diffusion formulæ (5) or (6) may presumably be used in calculation. When the duration of the train is greater than this, the diffusion formula may not be used. There is no available theory of mechanism in this range, unless the descriptive or empirical theory of Almeida¹² may be so classed. Stimuli of such

t	Vt (a)	(C)	(B)	(D)	(E)
0	0	0	0	0	0
.05	.2236	.00745	.2161	.0075
.10	.3162	.02108	.2955	.0207
.20	.4472	.05963	.3921	.0551
.30	.5477	.1095	.4502	.0975
.40	.6325	.1687	.4879	.1446
.50	.7071	.2357	.5125	.1946
.60	.7746	.3098	.5279	.2465
.70	.8367	.3905	.5367	.3000
.80	.8944	.4770	.5405	.3539
.90	.9487	.5692	.5407	.4080
1.00	1.0000	.6667	.5381	.4619	.785
1.20	1.0955	.8764	.5269	.5686
1.40	1.1832	1.1043	.5108	.6724
1.60	1.2649	1.3492	.4922	.7727
1.80	1.3416	1.6099	.4725	.8691
2.0	1.4142	1.8856	.4525	.9617	1.620
2.5	1.5811	2.6352	.4053	1.1758	2.360
3.0	1.7321	3.4642	.3641	1.3680

intensity and form as to fail to excite in 3 or 4 chronaxie may either depress the tissue to a subnormal state or may supersensitize it. The nature of the stimulus and the previous history of the tissue determines the course of this adaptation or accommodation.

The data of principal interest in this connection are those of Chauchard¹³ on latent addition. This is work from Professor Lapicque's laboratory at the Sorbonne. The stimulus is applied with Lapicque's usual type of electrode and consists of a series of identical relaxation impulses of time constants which are short compared with the chronaxie and spaced at equal intervals which are long compared with the chronaxie. A fixed number and spacing may be chosen and then the current or voltage adjusted until the complete train just excites the tissue. Experiments were usually done on the columella muscle of a snail.

The logarithmic plot, Fig. 4, represents two sets of data taken on one preparation (experiment of January 15th). The number of impulses is 12. One of these runs was taken with an impulse of effective length .0037 chronaxie or .000074 seconds, and the other .0093 chronaxie or .000185 seconds, approximately. Durations are taken to be the time constants, the error involved being trivial for this purpose. The abscissæ represent total duration of the stimulus in seconds on the logarithmic scale and the ordi-

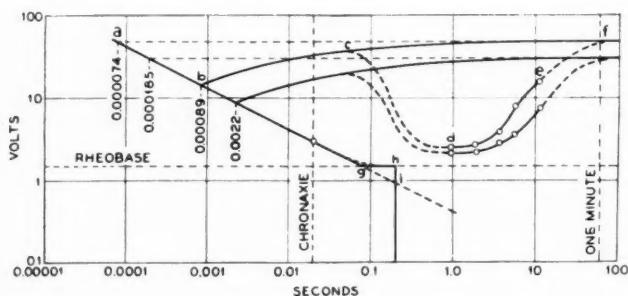


Fig. 4.

nates, the exciting voltage, also on a logarithmic scale. The data for the shorter impulse are given on the part of the curve lettered d-e, extending from a duration time for the 12 pulses of from about .9 seconds to about 11 seconds. As the impulses are separated, varying the duration of the train from the lowest value up to about 1.5 or 2.0 seconds, there is little change necessary in the intensity of the stimulus to effect excitation, the value being about 2.5 volts. As the separating interval is further increased the necessary intensity of the pulses increases more rapidly so that when the duration of the train is 11 seconds something over 15 volts is required. This represents the data for this run.

The value of chronaxie given for this sample of tissue is .020 seconds and the rheobase 1.5 volts. These two figures permit of the reconstruction of the rectangular impulse excitation curve a-b-g-h in accordance with the reasoning given in section 2. From this curve it may be seen that a single impulse, assumed now to be rectangular and having the duration as calculated above of .000074 seconds, would require about 48 volts to excite. This point is marked a on the single pulse curve. Assuming then that the 12 pulses are rectangular and contiguous, a single rectangular impulse of duration about .00089 seconds results. This, according to the curve, would require about 15 volts to excite and the point corresponding is marked b on the curve.

Imagine these 12 pulses to be then gradually separated, forming a succession or train, and the diffusion phase to be indefinitely long. The excitation curve is then b-c-f and is given by the series

formula (5), which is preferably modified for convenience for the particular purpose in hand. Consider the duration of each pulse to be δ and the time between the beginning of one pulse and the beginning of the next—that is, the periodicity, to be τ . If there are n such pulses so that the total duration time $t = (n-1)\tau + \delta$

$$\frac{y}{i} = [\sqrt{(n-1)\tau + \delta} - \sqrt{(n-1)\tau}] + [\sqrt{(n-2)\tau + \delta} - \sqrt{(n-2)\tau}] + \dots + [\sqrt{(n-(n-1)\tau + \delta} - \sqrt{(n-(n-1)\tau)}] + [\sqrt{(n-n)\tau + \delta} - \sqrt{(n-n)\tau}] \quad (15)$$

The last term corresponding to the last or n th pulse, is, of course, simply $\sqrt{\delta}$.

A simple point to calculate is that for which the separation between pulses is equal to their duration—that is, $\tau = 2\delta$.

$$\frac{y}{i} = \sqrt{\delta}[1 - \sqrt{2} + \sqrt{3} - \sqrt{4} \dots + \sqrt{2n-1} - \sqrt{2n-2}] \quad (16)$$

For $n = 12$, as in this particular experiment, $y/i = 2.80 \sqrt{\delta}$.

A convenient asymptotic value for the condition where the spacing is small compared with duration may be easily obtained, but as it will be seen later this is unnecessary here. The asymptotic value of (15) for the condition where δ is negligibly small compared with τ is desirable. By factoring out the first term $\sqrt{(n-1)\tau}$ from the second term $\sqrt{(n-2)\tau}$, and so on, and then extracting the square root of the remaining portion of each (15), reduces to the asymptotic form

$$\frac{y}{i} = \sqrt{\delta} + \frac{\delta}{2\sqrt{\tau}} \left[1 + \frac{1}{\sqrt{2}} + \frac{1}{\sqrt{3}} + \dots + \frac{1}{\sqrt{n-1}} \right] \quad (17)$$

For this experiment where $n = 12$, the series has a value of 5.32, so that

$$\frac{y}{i} = \sqrt{\delta} + 2.66 \frac{\delta}{\sqrt{\tau}} \quad (18)$$

From this information the curve b-c-f was calculated. It will be seen that this would represent the excitation curve for Chau-

chard's series of pulses if the normal or diffusion phase of this tissue persisted indefinitely. The actual data fall far below this curve.

From previous experience with the single pulse data it was concluded that the diffusion phase persisted only up to about 3 chronaxie. It may be assumed, tentatively at least, that if Chaudhary had begun the tests with the pulses contiguous, and then gradually separated them he would have proceeded along the diffusion curve, beginning at the point b, up to the point c, at which latter point the total duration of the train would have been about .06 seconds, or 3 chronaxie. Filling in the gap between the point c and .9 seconds, where the data begin, must be done by guess. This is done in the figure by drawing a smooth dotted curve descending from the diffusion line to the data curve, but there is no reason why it might not first rise and then drop to the data line.

In carrying the curve beyond the time intervals measured, the trend of the curve indicates a direction for extrapolation. The point in time at which this extrapolation reaches 48 volts corresponds to that at which the first impulse of the train alone will excite. The extrapolation, as drawn, suggests this point to be at about one minute, which would then be interpreted as the longest possible time for "summation" or "addition" to be a factor for this tissue with 12 impulses of the length used in the test. This time, being found by extrapolation, is, of course, very inaccurate but it indicates that a time of the order of magnitude of 5 seconds or 250 chronaxie is required by the tissue to completely recover from the effects of a single subthreshold pulse of the size used in this experiment. The total effective time for excitation might easily be as much as 10 minutes or longer and may possibly be associated with the protracted oxidative recovery time (minutes) of Gerard Hill and Zotterman.¹⁴ Similar curves plotted from other data given in the same paper indicate much longer times, particularly for a larger number of impulses.

From the above description it will be seen that the complete "addition" should be b-c-d-e-f for 12 pulses, each of duration .000074 seconds. The significance of the lower curve represent-

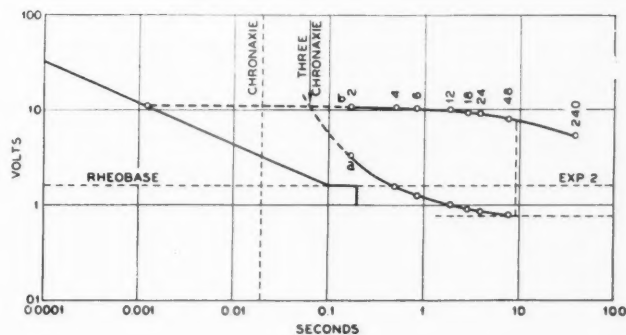


Fig. 5.

ing a similar run also with 12 impulses, but each of duration .000185 seconds, will be obvious.*

Fig. 5 represents a series of similar data taken in a slightly different way. In this case the time interval between impulses was kept constant at $1/6$ seconds and the number varied from 1 to 48, as indicated by the numbers. Total effective time of any train is plotted as abscissæ and the exciting voltage as ordinates, both on logarithmic scales. In this particular run a figure is given for chronaxie as .020 seconds and the rheobase as 1.6 volts, from

*Since the time of presentation of this paper Prof. H. W. Davis has generously read my manuscript and given his comments informally. Since his views are critically pertinent to the interpretation of the Chauchard data here used by illustration I venture to mention them here for the benefit of any reader who may not have access to the original discussion to be printed—no doubt in full—in the 1932 Transactions of the Otological Society. Consultation of the original is advisable. Prof. Davis is inclined toward the view that "The greater effect of a series of impulses is due to summation of excitation at the nerve endings—or more probably causing a sufficient contraction to be visible," that is, that the observed threshold is that of the eye of the observer and not of the muscle preparation. "Stronger stimuli excite more fibres, therefore fewer stimuli are needed for visible contraction." . . . "Personally I have practically abandoned the idea of 'latent addition' in such tissues as this, although summation in the central nervous system is still apparently a real phenomenon." This brings into the question the propriety of using these particular data for quantitative determination of magnitude, trend and duration of metabolic adaptation. As pointed out later there is another species of reasons for presuming that these data are inadequate to uniquely determine the mechanism responsible for the observed phenomenon.

which the single rectangular pulse excitation curve was constructed as before. The length of the single impulse is given as approximately $.55/10$ chronaxie, or about $.0011$ seconds. The voltage given for the single impulse is 11. This voltage when referred to the single impulse curve indicates $.0012$ seconds, so there is fair agreement between two methods of calculating the impulse length in actual seconds. The data are represented by the curve a and indicate that as the number of impulses are increased, keeping the interval constant, the exciting voltage falls to an asymptotic value of about $.76$ volts when the duration of the train is about 10 seconds, corresponding to about 50 or 60 pulses. In order to get the curve representing the "normal" or diffusion phase of the tissue, formula (5) was used and the curve b obtained. It will be seen that this gives a picture to be expected

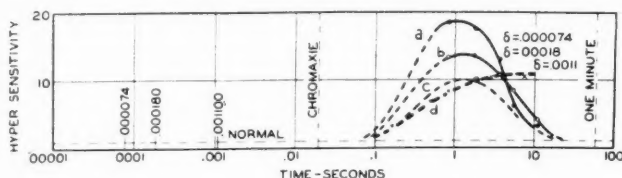


Fig. 6.

from the data previously considered in that the tissue is induced by such stimulus to augment its own sensitivity by a factor of 10. There are other data given in the paper which cannot be used in a study of this kind because, owing apparently to a large drift during the experiment in condition of the tissue, they lead to conflicting single impulse curves.

Fig. 6 is a plot of the degree of hypersensitivity taken from Figs. 4 and 5 of the tissue as a function of excitation time. Curves a and b are taken directly from Fig. 4 and indicate that the maximum of hypersensitivity occurs in the neighborhood of one second for the 12 impulses and that its value is a matter of 20 times for impulse durations of $.000074$ and 14 times for impulse durations $2\frac{1}{2}$ times as long. Curve d is obtained from Fig. 5, showing the hypersensitivity for impulses of rather long duration, $.00011$ seconds, as the number of impulses is varied. The maximum in this case is about 11 times, which occurs when the number of impulses

exceed about 20. The single point indicated by a cross surrounded with a circle is the one point on this curve corresponding to 12 impulses. This point must, therefore, be one of the points on a curve similar to a and b for a longer impulse than either of the other two and is indicated provisionally by the dotted curve c.

It is interesting to note that the time constants, if this be a proper term of chemical reactions in the tissue, are of such an order of magnitude as to have their maximum effect at about 50 chronaxie—that is, one second in this tissue.

Taking these curves as characteristic and disregarding for the present any inaccuracies which must necessarily enter into their makeup, there are a number of useful conclusions to be drawn. If the diffusion phase be regarded as the normal or resting phase of the tissue, it is evident that when the total duration time of the train is in the neighborhood of one second—that is, in the range of 25 to 100 chronaxie, the tissue is in a highly supernormal or hypersensitive condition. These curves indicate that when the impulses are normally subthreshold by a factor 15 to 20, they induce, by virtue of protracted intervals between them, a very much changed metabolic rate and induce an activity all out of proportion to the physical stimulus. The excitation of the tissue is thus primarily not produced by the physical process of "summation" of stimulus, but rather—the stimulus acts principally to condition the metabolic process (catalysis ?) in such a way as practically to induce it to excite itself. Within the diffusion phase there is, according to present conceptions, a true physical addition or summation of stimuli assumed to be represented approximately by the series (5) or (6), but outside this phase, in particular in the Chauchard range, the term "addition" is not concisely descriptive of the phenomenon.

The curves of Fig. 6 suggest that a train of a given number of impulses produce higher supersensitivity when these impulses are shorter within the range of impulse length measured, but other data are not in accord with this conclusion. The question must be left open for the present.

In contrast with the supernormal or superdiffusion condition induced by these small pulses, the single impulse longer than three or four chronaxie results in a subnormal or subdiffusion phase.

It is as though, while the stimulus physically tends to activate, it also induces a contrary tendency in the metabolic process while it lasts and a slow activating process lasting for a time after its disappearance. This matter is discussed by Almeida.

It might be of interest to note in passing that the supernormal phase of Keith Lucas is not related to that described here in any simple way. His supersensitivity occurs under certain circumstances after an excitation has taken place, whereas that of "summation" is built up before excitation. In any case Keith Lucas' supernormality amounts to only 5 or 10 per cent, which is very small compared to that in latent addition.

The value of any determined ratio of hypersensitivity as plotted, for example, in Fig. 6, is dependent on the accuracy of the data and on that of the theoretical or normal diffusion curve. The theoretical curves are spaced in voltage a distance proportional to the square root of the time interval of duration.* This is $\sqrt{5/2}$ in Fig. 4. If it had happened that the data showed a similar diffusion spacing, then the hypersensitivity ratio obtained would have been the same for both these experiments. This raises the question as to how nearly these curves are representative of the data in this respect. More specifically, it is desirable to know just how the levels of exciting voltage are related to time intervals of the pulses in the Chauchard range.

There are in the data figures on a number of experiments performed with different time intervals on the same tissue preparation, taken on the same day, and presumably with precautions to keep the preparations in as constant a metabolic state as possible. The additional data agree in two particulars, namely, that shorter impulses require higher voltages for summation in the same time, and that higher voltages are required for longer duration time of a complete succession when this duration exceeds one or two seconds. If all these curves are plotted, there is no quantitative law in evidence. In some cases the curves are closer together at one end; in others, closer together at the other end.

*Lapicque has for a time favored a type of relaxation hypothesis for the physical component of excitation. Figures of hypersensitivity based on this theory give greater values. This hypothesis seems now to have been discarded.

Some are farther apart at the middle of their course and others at the ends. There is no characteristic shape for the curves aside from the one mentioned of an upward trend after one or two seconds. This suggests that the excitability of the tissue was continuously in a random shifting condition during the process of any experiment, for causes unknown or not described by the author. This being the case, a statistical study is suggested.

When a single pulse excites within the diffusion phase the voltage-time relationship is the square root or diffusion law. It does not seem unreasonable as a trial hypothesis to assume that when a series widely spaced of subthreshold pulses act, each individually polarizes the membrane according to the diffusion law and is physically dissipated before the incidence of the next pulse, but initiates a process of slow auto-activation. Each pulse might thus be considered as falling within the diffusion phase of a tissue which is gradually shifting its threshold during the process of stimulation. As an additional part of the trial hypothesis assume the permeability of the cell surface to one ion to remain negligible throughout except at the instant of excitation and also that the diffusion and all other physical "constants" do not undergo variation. To test this out on the first point of the data of Fig. 4, the voltages given are 2.2 and 2.5 and the times in the ratio 5:2. If the diffusion law held as above described, the ratio 2.2:2.5 should be $\sqrt{2:5}$, or 2:5 to the power .50. This ratio 2.2:2.5 is, however, equal to 2:5 to the power .14, which is discouraging. A similar calculation taken from 99 points of these data shows this particular value to be unusual and gives an average exponent or power of .656 with a "standard" deviation of .22. Fig. 7 is a

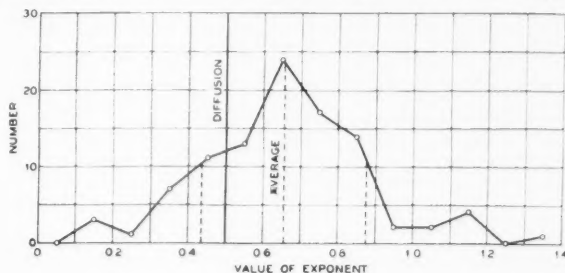


Fig. 7.

frequency of occurrence plot of the exponent counted in intervals of .10. The spread of the values is so great and the number of readings so small that the prospect of deducing any definite law is rather unsatisfactory. However, the diffusion law corresponding to .50 is not impossible. The excess .156, of the average exponent over that required for simple diffusion, suggests that other trend factors enter the phenomenon. A gradual increase in permeability of the cell surface, a decrease in the metabolic rate and a thickness effect might be among these factors. Other factors, not under control of the experimenter, enter into the phenomenon at random, which account for the large dispersion. It is concluded that the value of calculated hypersensitivity in the metabolic phase is quantitatively unsatisfactory but that a value of 10 at maximum is the probable order of magnitude.

It is to be hoped that the Paris laboratory will find means of measuring the complete curve from contiguity of the component pulses up to the end of the summation time, preferably with rectangular waves, and to find means of eliminating or determining quantitatively the drift in tissue condition which vitiates to a degree the utility of these measurements. Figures on rheobase and on chronaxie in seconds are necessary in each case in order to determine hypersensitivity. If the diffusion phase is as important as here assumed in determining the reference or "normal" properties of tissue, then it would be advantageous to physiologists to standardize, if possible, on methods of measuring rheobase and chronaxie—that is, to adopt some form of pulse, such as the rectangular, and some definite form of electrode. All data then taken on excitation would be comparable.

OVER-ALL MEASUREMENTS ON THE HEARING ORGAN.

In the interpretation in the light of what has preceded of the physical data to be presented, there are many difficulties which do not exist in the complex but comparatively simple (and as yet not quantitatively known) phenomena of tissue excitation. When a threshold of sensation is reached, an excitation is accomplished of each of a succession of elements, which form an unbroken path in the network of a sensory nerve. It is hoped, however, that sufficient progress will be shown to demonstrate the advantage of theoretically guided experimentation. A large proportion of

the precise data on hearing has been obtained largely for the sake of direct utility in the design of various acoustic appliances. These data are usually in the form of functional averages which partly, and sometimes wholly, conceal the details of the underlying mechanism. Many of these data are applicable and indispensable to the more fundamental problem of mechanism. However, they must be supplemented by additional measurements planned with discrimination.

1. *Absolute Acuity for Pure Tones.*—Data on acuity or absolute pressure sensitivity of the ear taken for use in this paper¹⁵ are plotted in Fig. 8 as a function of frequency. The pressures are given in the usual logarithmic units or "db" relative to 1 dyne/cm.² 20 db corresponding to a ratio of pressure 1:10. The data taken for the particular purpose discussed in this paper ranges from 35 to 1000 cycles. Measurements were made on 10 individuals of normal hearing at 5-cycle intervals from 35 to 100 cycles, and at larger intervals from 100 to 1000 cycles as indicated. These measurements are plotted as heavy dots, which represent average values. The mean deviation range is indicated by vertical lines. Before presenting the average curve, the individual curves were plotted, and it was found that the variations between ears are rather small and, particularly, that the trends as a function of frequency are very nearly alike. Each is represented by a straight line at low frequencies having the same slope as that drawn in Fig. 8 to represent the average, but displaced either up or down by an amount suggested by the deviations plotted. This slope at low frequencies is taken to be an invariable characteristic of the ear.

The method of making these measurements consisted in effect in measuring the threshold current in an electromagnetic receiver small enough to be inserted into the ear canal and sealed there with vaselin. This receiver was calibrated by inserting into an air chamber of volume equal to that of the average ear, one wall of the chamber consisting of the diaphragm of a small condenser transmitter which had previously been calibrated by well known methods. A determination was then experimentally made of the pressure developed in the air chamber resulting from measured currents in the receiver, as a function of frequency, and the

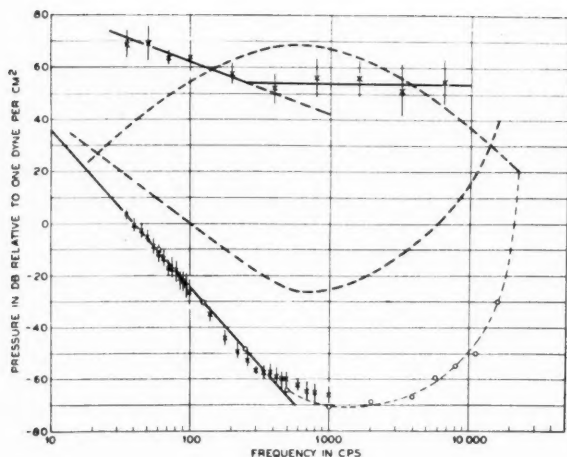


Fig. 8.

assumption made that the pressure developed in the ear canal was the same as this for the same exciting current.

This method involves the possible error, as has been pointed out on a previous occasion,²¹ that a yield of the ear drum relieves the pressure somewhat, whereas the stiffness of the diaphragm of the condenser transmitter is so high that yield is inappreciable. At frequencies below a certain value this error is necessarily constant but does not affect the slope of the curve, for the reaction of the ear drum must be purely elastic.

Three determinations by different methods of the impedance of the ear have been made,* and all agree that the impedance measured at the opening of the ear canal is preponderantly elastic at frequencies below at least 300 and possibly as high as 500 cycles. The stiffness measured is about $.1$ or $.2 \times 10^6$ dynes/cm², on a basis of canal area of $.45$ cm². Deductions from these figures

*The first, a crude determination, by Wegel and Lane, Bell Telephone Laboratories, 1923, unpublished, for this particular purpose soon after the question was raised gave stiffness of $.14 \times 10^6$; a second and much more accurate measurement by A. L. Thuras, Bell Telephone Laboratories, 1925, unpublished, in connection with the efficient design of certain telephone receivers, gave stiffness $.19 \times 10^6$, and a third by J. Troger, *Phys. Zeit.*, 31, 26, 1930, gave $.15 \times 10^6$.

indicate the stiffness measured at the drum itself to be something like two or three times this value.

An independent determination of the hearing threshold¹⁶ made on seven ears by measuring the pressure directly in the ear canal when a sound of threshold value is incident on the ear. The apparatus, which is considerably more elaborate than that described above, consisted essentially of a small tube inserted in the ear canal in such a way as to permit sound to enter and then attaching a condenser transmitter at the other end of the tube so that the pressure was measured directly. Attenuation of sound in this tube was, of course, corrected for. The values thus determined are plotted as circles in Fig. 8, and it will be seen that at low frequencies they are scarcely distinguishable from those determined by the first method.

The absolute values are given because of their probable value elsewhere but are of no consequence here. It will be seen that for low frequencies the straight line represents the data quite well, passing almost exactly through the Munson points up to 500 cycles, and through the others with the same accuracy up to at least 100 cycles and probably up to 400 or 500 cycles. The slope of this line is such that the threshold pressure drops 60 db or changes in the ratio 1:1000 in a range of frequency 1:10. Its equation is therefore given by the inverse cube law:

$$p = \left(\frac{40}{f} \right)^3 \quad (19)$$

The significance of such a low frequency trend, which is discussed in considerably more detail in the reference, is substantially as follows:

The impedance of every element except the fluid canals of the ear mechanism is predominantly elastic below some frequency—unspecified for the present. The helicotrema is a pure dissipative resistance at such low frequencies. Regardless of the nature of the relative motion of the parts of the measuring receiver, ear drum, ossicles, basilar membrane, round window or associated air chambers, or of their relative magnitudes, it may be demonstrated by a simple principle in mechanics that the whole network performs like a very simple system in so far as concerns the

motion of the receiver diaphragm, the motion of the basilar membrane, and the flow of fluid in the helicotrema. This principle has nothing to do with any particular "theory" of hearing. The whole apparatus, including the receiver diaphragm but excluding the basilar membrane and helicotrema, may be replaced by a single elasticity of the proper value, which is quantitatively immaterial here. The basilar membrane, moving slowly up and down, may be considered as a single elastic member in shunt with the dissipative resistance to fluid flow in the helicotrema. For such slow motions the shape of displacement of the basilar membrane does not change appreciably with frequency and all parts move in phase. Reckoned from zero of frequency any deviation from type of motion is presumed to be of second or higher order, but this remains to be checked theoretically and the point reconsidered if a first order change is found. With an unvarying motion, such as that assumed, it may be easily demonstrated that if a vibrating force acting on the receiver diaphragm be held constant in amplitude, as the frequency is changed the amplitude of displacement of the basilar membrane is proportional to the frequency. If, however, the force on the receiver diaphragm, to which the pressure in the ear canal is proportional, be made to vary inversely as the cube of the frequency, as for instance, in equation 19, then the amplitude of the motion of the basilar membrane is proportional to the inverse square of the frequency, its velocity inversely proportional to the frequency, and its acceleration invariable with frequency.

The measurements plotted in Fig. 8 were made as low as 35 cycles, and if it be conceded that unlimited extrapolation to lower frequencies is justified, this is an experimental demonstration that below a certain frequency the threshold of hearing is attained by adjusting the amplitude of vibration of the basilar membrane so that the acceleration is constant as the frequency is varied, or that for the threshold the acceleration of the basilar membrane is the direct stimulant. This was suggested on intuitive grounds by Roaf.¹⁷

The "low frequency range," as here understood, is that bounded on its lower side by very slow fluctuations, "zero" frequency, and at its upper side by that frequency below which the basilar

membrane moves substantially as a whole—that is, all parts “sensibly” in synchronism. It will be obvious that if a low enough frequency be chosen, say for safety, one vibration in ten seconds, .10 cycles, the basilar membrane must move as a whole. The same will then necessarily be true for one vibration in 100 seconds, or .01 cycles per second. It will also be obvious that as the frequency is gradually increased from such a low value, the shape of vibration of the basilar membrane, rigorously considered, undergoes gradual change. The question as to designation of the frequency at which this change in form of vibration becomes “sensible” or perceptible has not been answered. It is provisionally identified here with the place at which the deviation from the inverse cube law for acuity takes place, namely, between 100 and 500 cycles. No sufficient proof of this is offered, and it is subject to revision, probably downward, when further pertinent information is forthcoming.*

In this slow frequency range the ear behaves mechanically as a purely elastic device, as it has been supposed in some of the earlier conceptions of the mechanism of this end organ. The acceptance of this conclusion implies that it is also agreed that all discrimination as to pitch and intensity is a function exclusively of the properties of the nervous tissue of the auditory tract. It is also implied, if the tension and thickness of the basilar membrane be supposed to be about constant along its length, that up to the frequency defining the limit of this low frequency range the deflection of the basilar membrane at any point, except in the neighborhood of its ends, is approximately proportional to its width. If the precision measurements of Guild on guinea pigs¹⁸ may be taken as indicative of the nature of variation of width of the basilar membrane in man, then the low frequency deflection increases rapidly from the proximal end for a distance of about one-third its length, and then increases more slowly for the remainder of the distance with a few mild superposed undulations of a matter of 5 per cent of the main deflection.

*Since going to press, time has permitted of a rough calculation which indicates that the mechanical “low frequency range,” as it applies to the cochlear galleries, is probably below 35 cycles, and that, therefore, definite conclusions regarding the nature of the stimulus as here described are best held in abeyance pending a more complete investigation.

If at low frequencies the threshold is attained by a constant acceleration, this may not be safely presumed to hold for the higher frequencies. Nervous tissue excitation apparently does not follow a simple law, as the frequency of stimulation is varied over a wide range. However, if such be assumed provisionally to be the case over the entire range, then the surprising sensitivity of the ear at very high frequencies is plausibly explained. The mechanism of the ear does not have the appearance of a high frequency device. A relative mechanical efficiency of the ear (transfer impedance) would be obtained by multiplying the acuity curve of Fig. 8 by the square of the frequency. This is plotted in Fig. 8 and marked "b." The units are arbitrary. This curve shows the maximum of mechanical efficiency to be somewhere in the neighborhood of 1000 cycles, whereas that of maximum acuity is perhaps one to two octaves higher. The extreme high frequency sensitivity is thus largely due to the properties of nervous tissue.

It will also be obvious that it does not necessarily follow from the acceleration rule for threshold at low frequencies that acceleration is the physical stimulus for intensities higher than the threshold.

Acceleration is a mechanical term used here in the limited sense of describing a certain characteristic of a vibration. When the acceleration is constant in a simple vibration the required amplitude of motion is proportional to the square of the time interval between successive impulses of the sound wave; the velocity of motion is directly proportional to the time interval, and the acceleration of the motion is independent of it. For interpretation, reference must be made to tissue excitation data. Ideally, this should determine certain properties of the receptor hair cells, analogous with but distinct from those of the nerve tissue.

Most excitation data are threshold determinations. Thus the excitation at threshold of a nerve trunk is presumed to have taken place when a muscle twitch is observed by the eye, or when an action current is first observed by means of a galvanometer. There are many good reasons for concluding that the "threshold" is not an experimental artefact. In hearing measurements it is also assumed that a threshold exists in the complete organ; that it corresponds to that stimulating intensity which is just sensible,

and that the intensity of this stimulus at the nerve terminal is the same as that which would be there measured instrumentally after making due allowance for differences in physical application of stimulus by receptors and by electrodes.

It would be fortunate if types or forms of mechanical stimuli could be applied to a fixed number of receptors of the organ of Corti identical with the electrical pulses used in obtaining the tissue excitation data previously described. Thus it would be convenient if the basilar membrane could be bodily displaced and then returned to its normal position after a measured interval for the purpose of giving it a "rectangular" mechanical impulse through its resection on the fluid in the scala media or on the membrana tectoria. This might be compared with the same type of electrical impulse applied directly to the nerve and the separate properties of the receptors deduced. When, however, such a rectangular pulse is applied in the form of sound at the external ear it is mechanically warped, distorted and dispersed over the membrane in such a way that the displacement is nowhere a copy of the impressed stimulus. The nearest approach to correspondence between direct stimulation of the receptors and an externally applied disturbance is to be had with a sustained pure tone. In this case the tissue receives periodic stimuli having the period of the tone but unfortunately with greater violence in some regions than in others. In this respect it is not greatly different from the conditions obtained in electrically stimulating a nerve trunk in which only a limited number of fibers in favorable positions are excited.

When a pure tone of low intensity and of sufficiently low frequency acts on the ear, the basilar membrane moves up and down with all its points substantially in synchronism, since the diameters of the canals are much larger than the width of the basilar membrane. The limiting frequency at which this simple vibration may be supposed to take place has already been suggested to be between 100 and 500 cycles.

The Wever and Bray experiment has indicated that in this, as well as in much higher ranges of frequency, the periodicity of the generated composite impulses in the nerve trunk is the same as that of the stimulus. It has not been demonstrated that

at the threshold each fiber concerned is excited once for each period of the sound. In the normal ear the continued vibration at threshold value probably induces in the tissue a metabolic or extra-resting phase.

Before passing to a consideration of the excitation mechanism it may be helpful to notice that the interval between successive impulses in a 35 cycle wave is .03 seconds and that the shortest interval in the "low frequency" range is in the neighborhood of .003 seconds. The diffuse electrode chronaxie of the auditory nerve trunk is probably about .0001 seconds. The sine wave stimulus is the smoothest varying and hence the least efficient periodic stimulus.

As the fibers of the auditory nerve (see Fig. 9) enter the scala media through the foramina in the lamina spiralis they lose their medullary sheath. From this point they project to the nonciliated ends of the hair cells and turn at right angles, forming bundles or bands, one of each of which follows the spiral course of a row of hair cells. The diagrams of Retzius suggest that one fiber may extend along a spiral band a distance of 20 or more hair cells and that it may branch a number of times. The fibers terminate by arborizing about the ends of the hair cells.*

A conceptual picture of what appears to be a plausible mechanism for transformation of the mechanical stimulus into a nerve impulse may be had by considering a single hair cell with a single fiber terminated near its lower end. It is not proposed, of course, that these units actually act independently of one another. When vibration takes place there is in general a motion of the organ of Corti normal to the plane of the basilar membrane and a synchronized longitudinal motion of the fluid of the scala media. The result of these motions is to apply a shearing stress on the cilia of the hair cells and a consequent extensional strain on some excitable interface on or near the cilia. An extension of such a surface results in the attenuation of its charge and a tendency to activation similar to that produced by passing a current through it. The amount of the extension is proportional to the electrical charge necessary to pass through it to produce the same degree

*Recent investigations of de No have profoundly modified this picture but not in such a way as to materially affect the present conclusions.

of activation, and the velocity of the extension is at any time proportional to the equivalent current density required to produce the same rate of activation. In this way it may be seen that the current as a stimulus is interchangeable with velocity of extension of an excitable surface or, what here comes to the same thing, with velocity of motion of the basilar membrane. A cathodal stimulus corresponds to a motion producing extension of the excitable surface of the ciliated end of the hair cell and an anodal stimulus corresponds to its contraction.

The data and conclusion described under "excitation" are unfortunately not quantitatively applicable to excitation of the hair cells. The seat of excitation here is a very small area of the cell and should be quantitatively more nearly related to measurements made with "pore" or micro-electrodes. Chronaxies measured with these small electrodes may be as small as one-tenth¹⁹ those measured by the large electrodes for reasons which are not unexpected. The treatment of excitation under these conditions offers an attractive theoretical problem.

After excitation of the hair cell the activating or action "wave" is quickly propagated over its surface to its nonciliated end. At this point the surface is specialized and in close proximity with the fiber terminals. This junction point resembles a synapse. It may be supposed that here the ionic atmosphere of the two cells overlap, without metabolic contact, however, to such an extent that a sudden activation of the hair cell surface constitutes a stimulus for the fiber and after a certain time delay, corresponding in a way to a micro-electrode excitation time for the fiber, produces an excitation of the fiber which is in turn propagated centrally.

While it is convenient to think of velocity of extension or contraction of an excitable surface as corresponding to electrical cathodal and anodal stimulation, respectively, it should be borne in mind that for the purpose of quantitative mathematical analysis this concept is not sufficiently precise. An electrical stimulus, whether from a large or from a micro-electrode, is applied from without so that potentials are effective over a large area due to spread of the flow lines of stimulating current. This is never exactly a local effect. A strain in the surface defining the electro-

lytic double layer is, however, substantially local, being confined to the strained area and having a stray effect from such area at distances comparable only with the equivalent thickness of this double layer. By way of contrast, this is effectively an internal stimulation. The radius of curvature of surface elements in question is no doubt comparable with the depth of penetration of diffusion when the stimulus frequency is low, so that a process analogous with Hill's thickness effect comes into play. If a cell or part of it is flexed so as to produce extension of its surface at one point and contraction at another, the stimulus is similar to that obtained with two micro-electrodes, one being a cathode and the other an anode. With these matters in mind, the obvious allowances for the qualitative description given here will be assumed to have been made without further explicit mention.

We thus arrive at the conclusion that the hearing threshold for sinusoidal stimulus is that at which a simple periodic current will excite and that the amplitude of this current is inversely proportional to frequency. If the hair cells and fibers are "isochronous," in Lapique's sense, the threshold is that of the hair cells, and the law of their excitation is not the unidimensional diffusion law of Nernst, for this law predicts velocity (current) to be inversely proportional to the square root of the frequency. If the rule of "heterochronism" prevails then the threshold corresponds to that of the fibers and their excitation at threshold is accomplished in a metabolic rather than in a physical phase by a process similar to that of "latent addition." In either case there are theoretical questions to be dealt with before any satisfying answer can be given.

As a matter of passing interest, attention is directed to the topmost curve of Fig. 8. This represents a new determination of the "feeling" threshold for the ear. At each frequency, as indicated by the points, the observer was asked to set the intensity first for the auditory threshold and then to increase the sound level until a second threshold was reached. This is usually described as "feeling," but its nature varies considerably with frequency and somewhat with observers. At lower frequencies a gentle but definite vibration is experienced which is quite distinct and superimposed on the sound. In some cases, however, "dizzi-

ness" is described, suggesting excitation in the semicircular canals. At higher frequencies the sensation is one of sharp pain.²⁰ It is not known which end organ or end organs are responsible for this threshold.

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LVIX.

CHEST COMPLICATIONS OF SINUS DISEASE.

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DALLAS.

For the past fifteen years there has been a growing realization among internists and otolaryngologists that many of the chronic and subacute diseases of the chest are dependent upon chronic or subacute infections of the nasal accessory sinuses. At first there were only a few men interested in establishing the relationship between chest and sinus diseases, but as the years have passed along, more men have investigated the close association that exists between upper and lower respiratory infections. At present it is realized by most men of the medical world that there is a relationship between sinus and chest conditions in a fair number of instances, but even now there are comparatively few doctors who know how constant is this relationship. I believe that almost 100 per cent of all chronic tracheobronchitis, bronchitis, bronchiectasis and peribronchitis cases are directly dependent upon some type of chronic sinus disease, with the exception of specific types of lung infection, such as tuberculosis, syphilis of the lungs, pneumonia or lung abscesses resulting from foreign bodies of various sorts or the aspiration of infected tissue or material at the time of operation. Even when the lung disease occurs as a sequel of measles, whooping cough, scarlet fever or influenza, it is probable that a sinus disease exists in those cases where the trouble in the chest persists and is resistant to measures of relief directed to the chest itself.

In one large clinic for thoracic diseases a study was made of 1,510 consecutive cases of pulmonary diseases. Of this number, 166 cases were tuberculous and the remaining 1,344 were diagnosed as nontuberculous. The majority of these 1,344 cases were diagnosed as bronchitis, bronchiectasis and asthma. There were a few that did not receive a positive clinical diagnosis. A sum-

mary of the 1,344 cases showed that 464 of them had deviated septums, spurs and hypertrophied turbinates. In this group 51 patients had total nasal obstruction most of the time, while 413 cases had partial blockage and occasionally total blockage. In all these 464 cases there were very frequent colds, which meant that there were acute exacerbations at times of a chronic sinus disease.

Another group of 582 of these cases showed constant nasal discharge; in 105 of them there was demonstrated one or more of the sinuses to be definitely infected. In 427 cases out of 582 of this group there was a demonstrable postnasal discharge of varying character. The remaining 298 cases of the total 1,344 nontuberculous cases showed diseased tonsils, enlarged adenoids and granular pharynx. Undoubtedly many of the cases of granular pharynx were dependent upon a postnasal discharge originating in infected ethmoid cells. T. R. Smith, who reported this group, states as follows: "No condition of the chest should be finally diagnosed without a thorough search for the foci of infection in the head."

Chronic sinus disease must be looked upon as a constant menace to the lungs. Rist very aptly drew the comparison that exists between the respiratory and the urinary tract when he said that as cystitis is so frequently the consequence of renal infection, so is chest disease frequently the consequence of sinus disease.

It is my intention in the presentation of this subject to explain the various possible routes by which the infection in the sinuses can reach and involve the chest structures. It is probable that in some cases the infection has been carried to the chest by one route almost entirely, but in the greater number of cases I believe that the infection probably gets to the chest by a combination of routes.

It seems that the four following routes of infection are worthy of consideration: (1) Lymphohematogenous, (2) hematogenous, (3) direct aspiration of infectious material into the trachea and the bronchial tree—droplet infection, and (4) direct continuity of tissue.

The lymphohematogenous route of infection from the sinuses to the lungs has been demonstrated by some experiments made by Drs. W. V. Mullin and C. J. Ryder, who reported the results of their observations in 1921. They showed that by injecting a carbon and tubercle bacilli mixture into a sealed-off maxillary sinus of a rabbit, the particles of carbon were picked up by the lymphatics draining that cavity and were carried to the submaxillary and jugular lymph nodes. Sections of these glands showed the carbon to be in them. Some of the carbon passed through the deep cervical lymph nodes and found its way on the left side into the left jugular lymphatic trunk and thence into the thoracic duct that empties into the left subclavian vein. On the right side the route is the same except that the right jugular lymphatic trunk empties direct into the right subclavian vein. The carbon was then carried into the right auricle, thence into the right ventricle and was sent out into the pulmonary arteries. They made sections of the pulmonary arteries and lymph structures surrounding the bronchioles and found the carbon in the arteries and lymph structures. A tuberculous ulcer had developed in the mucosa of the injected antrum and this was thought to be the point of entrance of the carbon into the lymphatic vessels. This experiment made these men feel that probably this lymphohematogenous route was of first importance. They found that infections of the frontal sinus seemed to follow the same course of lymphatic absorption as from the antrum. I want to quote from the authors as follows: "Drainage of the sinuses into the upper air passages renders the bronchi and lung liable to infection by inhalation, and this factor may be as important as lymphatic metastases in accounting for the clinical association of sinus disease with bronchopulmonary disease, though in nondraining cases it seems to be excluded." Unquestionably, many of the infections reaching the lungs from the sinuses are carried in this way. I believe that in instances of hyperplastic sinusitis and in sinus disease where there is practically no discharge from its mucosa this route of infection is probably the most important and certainly the most likely one.

The hematogenous route has been given very little consideration by most observers, but it seems to me not unlikely that some

infection might reach the lungs in this way. There is a very free venous drainage from the sinuses, and these veins could first carry the infection into the sphenopalatine vein and into the pharyngeal veins, thence into the internal jugular. After the infection gets into the heart, it would be shot out into the lungs through the pulmonary arteries and would finally probably involve the lymph tissue surrounding the bronchioles and bronchi.

Surely many of the chest complications of sinus disease can be explained by the aspiration of infectious material into the trachea and bronchial tree. This is the so-called droplet type of infection. The mucopurulent or purulent material that is manufactured in the sinuses finds its way out of these cavities and into the nasopharynx. From there some of it drops through the larynx into the trachea and thence into the bronchi and bronchioles. The mucous membranes of these structures then become infected and begin to produce a mucopurulent discharge themselves. At first we have a tracheitis, then a tracheobronchitis, and later a rather extensive diffuse bronchitis. If the bronchitis persists long enough, there will be some dilatation and probably sacculation of the bronchi with the development of a true bronchiectasis. If the sinus infection is checked, the bronchitis will usually clear up rather promptly; but if a bronchiectasis has developed, the lung infection will probably persist to some extent, regardless of how perfectly the sinus infection has been eradicated. After clearing the patient of sinus infection, the bronchiectasis may not get worse and the condition may even improve somewhat, but a complete cure cannot be expected where there has been a sacculation of the bronchioles. Certainly no permanent improvement can be obtained in these cases of bronchitis so long as the sinus infection is constantly causing infectious material to drop into the bronchial tree. The lymph nodes surrounding the bronchi and bronchioles become involved because they drain the infected mucosa of these tubes. An X-ray picture will usually show a peribronchitis. The bronchial glands surrounding the bifurcation of the trachea are the largest normally, and when they become heavily infected they may enlarge sufficiently to compress and narrow the canal of the bronchi. In studying this aspiration or so-called "droplet infection," I have done some work that I

believe to be original, so far as I am able to determine from available literature.

In an article entitled "The Relationship of Sinusitis and Bronchiectasis," by Drs. Lester H. Quinn and Ovid O. Meyer of Madison, Wis., that appeared in the August (1929) issue of the *Archives of Otolaryngology*, these gentlemen explained the details of an experiment they had made to prove that aspiration from the upper respiratory tract does occur while a person is asleep. I will quote as follows from their report: "A small catheter, about four inches in length, was placed in one nostril. Care was taken that the tip of the catheter did not enter the glarynx. The catheter was held in position by adhesive tape attached to the nose and face. During the night, between 11 p. m. and 5 a. m., at irregular intervals, from 1 to 2 cc. of iodized oil was gently put into the catheter with a syringe, until 20 cc. had been administered. As the patient awakened on the first few attempts, $\frac{1}{8}$ grain of morphin was given hypodermically and an hour later 10 grains of sodium barbitol was given by mouth. At 8 a. m. a roentgenogram was taken and iodized oil was found in small quantities in both lower lobe bronchi." The iodized oil disappeared from the lungs after one week's time. Later they repeated the experiment when the patient was less apprehensive and did not use the sedatives. Again they recovered the iodized oil in the bronchi.

I decided that lipiodol should be recovered in the bronchial tree if it was injected directly into the sinuses instead of instilling it into the nasopharynx through a catheter inserted into the nose. I did not see why the lipiodol would not enter the trachea while the patient was awake. Accordingly, I injected the ethmoids and sphenoids with lipiodol, by the Proetz displacement method, at about 4 p. m. The patient was sent directly to the X-ray room and pictures of the sinuses were made. The lipiodol was in the ethmoids, sphenoids and right antrum, the left antrum having been almost completely obliterated by a Caldwell-Luc operation. The patient was told to avoid coughing and a sedative cough mixture, containing $\frac{1}{8}$ grain of codein per dose was prescribed to be taken every two or three hours if necessary to control coughing. The next morning a picture of the chest was made, but no lipiodol was

found in the bronchial tree. Twenty-four hours after the first lipiodol injection I repeated the same procedure by injecting some more lipiodol by the Proetz method. The patient went directly to the X-ray room again and pictures were taken of the chest. These pictures showed lipiodol in the bronchi. The lipiodol was not injected again, and twenty-four hours after the second injection, or forty-eight hours after the first injection, another X-ray picture was made, which showed the lipiodol had nearly all disappeared but there was still some of it scattered through the smaller bronchioles. Lipiodol is certainly more irritating to the tracheal mucosa than would be the mucopurulent discharge formed from the patient's own tissues; therefore, I thought that the value of the experiment would not be impaired by asking the patient to refrain from coughing, if possible, and I gave the sedative cough mixture to help in that respect. It is true that only a small quantity of lipiodol was found in the bronchial tree, but larger quantities of mucopurulent material manufactured from the patient's own tissue would probably be retained before it would be coughed out, because the lipiodol is a foreign material and is slightly irritating. I had also shown to my own satisfaction that the material could be aspirated into the trachea while the patient was awake. I think that unquestionably this experiment proves that aspiration infection plays a very considerable part in the production of these chest complications associated with sinus disease in which we do have a discharging type of infection. This does not mean that I am not fully aware of the frequency with which these infections are carried through the lymphatic-hemogenous route, but that I consider the aspiration type of infection also of paramount importance. (See Figs. Nos. 1, 2 and 3.)

The direct continuity of tissue method of infection should receive some consideration. Just as we see infections spread along a mucous membrane elsewhere in the body, in the same way an infection that would get to the larynx could spread down the mucosa of the trachea and along that of the bronchi and bronchioles. Infected material produced in the sinuses would drop from the postnasal space and infect the larynx and then spread downward by direct continuity of tissue, even though none of the infected material itself got into the trachea.

Spasmodic bronchial asthma is practically always associated with more or less disease of the sinuses. In 1913, Dundas-Grant found that in a series of 107 of his asthmatic patients there was a well marked nasal disease in 63 per cent and that where he operated for the relief of the nasal disease he got complete cure in 21 per cent of the cases, improvement in 60 per cent and no change in the asthmatic state in the remaining 19 per cent. In 1914, Mathews of the Mayo Clinic found that 90 per cent of a series of 300 asthmatic cases had sinus disease or some pathologic lesion in the nose and that relief of the asthmatic symptoms was in direct proportion to the thoroughness with which he was able to promote free and continuous drainage to the diseased sinuses.

Vasomotor rhinitis is associated with asthmas of all types and is usually a part of the asthmatic picture. It nearly always precedes an asthmatic attack. I believe that in nearly all asthmas there is one or more exciting agents or allergins, which may be pollens, animal emanations, food toxins, or bacterial toxins, but these agents are not the underlying cause of the asthma. In these cases the membranes are sensitized to action of the allergins, but the real problem to solve is just what has produced this sensitive state of the mucous membrane. Many observers, including Drs. Carmody, Green, Ferris Smith, Francis M. Rachemann and Harold G. Tobey, believe that infection is the principal factor in diminishing the normal resistance that these membranes have to such toxic agents. I share in this view of the matter and feel that there is always an infection of the sinuses in these cases and that its presence increases the permeability of the membranes to the action of the foreign protein. Drs. Carmody and Green called attention to the control of hay fever by draining diseased antra and cleaning up nasal infection. Very few otolaryngologists have not seen this happen, and many cases of asthma can be cured by completely ridding the patient of sinus disease. After a very exhaustive study of the subject, Dr. Ferris Smith concludes:

1. "A very large percentage of asthmas depend on chronic sinus disease.
2. "The asthma may result from a co-existing sinus disease and the presence of some extrinsic allergin.

3. "Many cases are not diagnosed because of inadequate methods of examination. Lipiodol injection and suffusion are of great service.

4. "Surgery in asthma cases has been discredited because of the poor conception of the involved pathology and insufficient surgery for its removal.

5. "In ethmoid infections, complicated with bone involvement, it is not possible to totally remove the disease in all cases. Infection may persist and polypi recur. All of the other sinuses may be totally cleared of infection.

6. "Only the most radical surgery will be adequate."

I feel that Dr. Smith is eminently correct in his observations, for my experience has many times borne out his conclusions. Several of my patients who were definitely sensitive to food toxins, to the extent that the ingestion of such foods would produce asthmatic attacks, have been desensitized to the action of these same foods by completely clearing them of some type of sinus disease. Sluder observed that he could frequently stop an asthmatic attack by cocaineization of the sphenopalatine ganglion. Surely sinus disease could easily produce a marked irritation of this ganglion.

Case No. 1.—Miss M. T. V., age 24 years. Sinuses were injected with lipiodol according to the Proetz displacement technic. The left antrum would not hold the lipiodol because a Caldwell-Luc operation had been done on that side and most of the cavity had been obliterated. Figs. 1 and 2 show the lipiodol in the sinuses, No. 1 being taken in the Granger position and No. 2 in the lateral position.

A chest picture was made fifteen hours after the lipiodol had been injected into the sinuses, but there was no evidence of opaque media in the bronchial tubes.

Another chest picture was made twenty-four hours after lipiodol injection of the sinuses, and it showed many fine globules of opaque media in the inner zones of both lung fields. There were many fine and large globules of lipiodol distributed throughout the lower portions of the bronchial tree, but it could be more distinctly seen in the right hylus region and right lower lobe.



Fig. 1 (Case 1). Showing lipiodol injected by Proetz method.
Left antrum almost obliterated by Caldwell-Luc operation.



Fig. 2 (Case 1). Lateral view showing lipiodol injection
by Proetz method.



Fig. 3 (Case 1). Lipiodol in the bronchial tree. Arrow shows a film of lipiodol in the right bronchus. This picture taken twenty-four hours after lipiodol was injected in the sinuses by Proetz method.

Fig. 3 shows the lipiodol in the bronchial tree. The arrow on the film points to a bronchus lined with a film of lipiodol. A chest picture was made forty-eight hours after the lipiodol injection, and by that time the lipiodol had lost its globular distribution and was rather diffusely scattered throughout the bronchial tree.

This case is not presented because of chest complications from sinus disease, though the patient actually had a fair amount of bronchitis with probably an early bronchiectasis. There was a chronic hyperplastic change in the left antrum with many polyps.

The Caldwell-Luc operation was done on that side and the anterior two-thirds of the left middle turbinate removed. A submucous resection had previously been done and the right antrum had been washed several times through a naso-antral window. The patient's chest condition has markedly improved since her nasal surgery.

The lipiodol injection of the sinuses was done to determine the possibility of secondary chest complications in just such cases, and I think that the study of the X-ray pictures after the lipiodol injection rather conclusively proves the frequency of such chest complications being explained on the basis of "droplet infection."

Case No. 2.—Miss J. E. L., age 30. Patient had pneumonia at 6, 12 and 23 years of age. Two attacks of influenza, and complained of spitting up blood each morning. Chest was negative on percussion. Whispered voice audible over both apices. Slight suppression of breath sounds in the right apex anteriorly. Slight cogwheel breathing in both bases posteriorly. Occasional coarse râle heard over bronchial area. Sputum is negative for tuberculosis. Diagnosis, by physical examination, is bronchiectasis.

Examination of the nose in September, 1931, showed that the anterior half of each middle turbinate had been removed. There was a heavy mucopurulent discharge draining from each ethmoid labyrinth that showed considerable degeneration. A bilateral ethmo-sphenoidectomy was done in November, 1931, and at this time the nose is apparently very clear on both sides. The post-nasal drainage has been stopped and the patient's general condition is considerably improved. The bronchiectasis has advanced too far to expect a complete recovery in that respect, even though the nasal infection has been cleared up. It may improve considerably, however, and she may not be troubled with it much in the future.

X-ray pictures of this case showed that the frontal sinuses were clear. Both maxillaries had some thickening of the lining membrane. The Granger line was considerably heavier than the average, and the lateral position showed a marked thickening of the lining membrane in each sphenoid sinus. The ethmoids also showed a marked thickening of the lining membrane throughout.

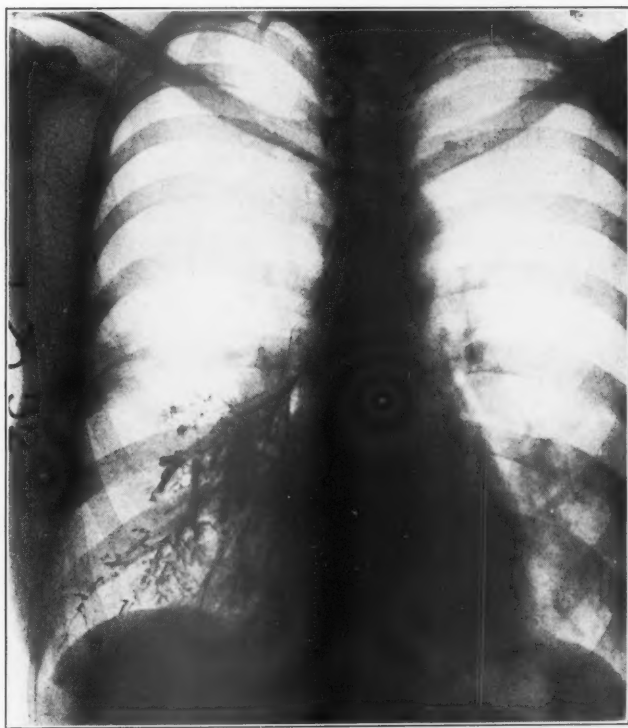


Fig. 4 (Case 2). Diagnosis, bronchiectasis. Right side of bronchial tree injected with lipiodol beautifully illustrating cylindrically dilated bronchioles with some tendency to sacculation.

Fig. 4 shows the right bronchial tree injected with lipiodol rather beautifully illustrating the cylindrically dilated bronchioles with some tendency to sacculation.

On May 5, 1932, approximately seven months after her nasal surgery, the patient stated that she rarely coughs and never raises anything from her chest. Her general condition is markedly improved. Before her operation her temperature would rise to 101 to 102 every afternoon. She has been free of fever for several months. In this case there had been too much dilatation of the

bronchioles to expect a return to the normal. However, as soon as the upper respiratory infection was improved, so that she no longer got a continuous dropping of mucopurulent material into her trachea, she showed a marked improvement in her general condition. This case simply illustrates the close association that exists between sinus infection and bronchiectasis.

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LX.

A REVIEW OF SINUS-CHEST INFECTIONS.*

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CLEVELAND.

It is now thirteen years since I first began the study of the relationship of sinus and chest infections. The study began with two sets of experiments performed on living animals, the first to determine the lymph drainage of the accessory nasal sinuses, and the second, the observation of experimental lesions of the lungs produced by the inhalation of fluids from the nose and throat.

I have studied a large number of clinical cases during these thirteen years, and a small group of them has been followed for the entire period—that is, from childhood to young adult life. The subject is kept ever before my mind, for I am seeing constantly patients of this type. During the past year, 1931, of 295 new patients with sinus disease seen at the Cleveland Clinic, almost 20 per cent of them complained of cough, while in one-third of these, or nearly 7 per cent of the whole group, cough was the only symptom.

The involvement in the chest is based on the infection and enlargement of the bronchial, peribronchial and mediastinal glands. This, in turn, produces irritation and cough, which if prolonged, interferes with bronchial peristalsis, and causes dilatation of the bronchi with retention of secretion.

Lymphatic absorption from the antrum, whether of bacteria or of inert substances, is by the way of the submaxillary and internal jugular nodes to the lymph ducts, the great veins, the heart and the lungs. Substances reaching the lungs may, of course, pass on to the general circulation.

My original lymph drainage experiments have been referred to frequently in the literature.¹ The inhalation experiments apparently have not been noticed equally, for they have been men-

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tioned far less frequently by other workers in the field. It is for this reason that I am repeating some of the details of those experiments which I feel have a special bearing on the present discussion.

I quote from a report which was published in the American Review of Tuberculosis in 1920.² "We found that light fluids, such as India ink (a suspension of minute particles of carbon), if allowed to flow into the nose, were very readily inhaled, producing effects in the lungs, the quick development and massive character of which, in the absence of any signs of absorption along the cervical or mesenteric route, gave convincing evidence that they were produced by pure inhalation.

This rapidity and massiveness seemed to give the method some advantages in a study of inhalation over the usual methods employing dust or spray, when the effects are produced more gradually, and it is not so easy to rule out absorption by other routes. We therefore performed a series of experiments by this method, in order to compare the effects thus produced with those found especially in pulmonary tuberculosis, and to observe whether there were any analogies which would constitute evidence for or against the importance of infection by inhalation.

The technic was as simple as possible. The rabbit was held, usually on its back, with the nose elevated, and about 1 cc. of india ink or bacterial emulsion was allowed to flow into one nostril from a pipette inserted just far enough to insure the fluid entering the nose. In some cases this was done several times, in others just once.

Rabbit 1 was given 1 cc. of india ink on December 17th. This was repeated eleven times in the next twenty-five days. Twenty-four hours after the last injection the rabbit was killed and examined. There was no discoloration detected in the upper air passages, trachea or cervical lymph nodes. The upper lobes of both lungs were almost entirely black, with many black spots irregularly distributed over the rest of the lungs. There was intense blackening of the bronchial nodes. Microscopic sections of the lungs showed carbon in the smaller bronchi and indicated that the blackening of the lungs was lobular in arrangement, the alveoli

being packed with carbon, much of which had been taken up by large phagocytic cells, evidently the alveolar cells of the lungs; and the deeper structural tissue of the lungs also contained many of these cells apparently traveling along the lymphatics.

The appearance of all this pigmentation, its overwhelming quantity, is absolutely distinct from spontaneous anthracosis, which, in rabbits, is seldom macroscopically visible in the lungs, and if so, occurs only as fine stippling, never as masses or blotches. Spontaneous anthracosis of the bronchial nodes is sometimes fairly marked, but never looks perfectly black.

The spleen showed a good deal of carbon microscopically in the sinusoids, and the walls of arteries in the kidney and elsewhere contained traces of it. There was no discoloration of the mesenteric nodes.

Rabbit 2 was given five injections of india ink over a period of a week and killed one hour after the last injection. Autopsy showed flecks of ink in the nasal fossæ, trachea and bronchi. There was massive blackening of the right upper and left lower lobes of the lungs, and deep discoloration of the bronchial nodes. The spleen contained a moderate amount of finely divided carbon. The cervical and mesenteric nodes were not discolored. Microscopic sections of the lung showed carbon packed in the alveoli, both free and in large mononuclear (alveolar) cells. The behavior of these cells in this instance, as well as their proliferation in acute and tuberculous pneumonia, emphasizes their close relationship to the fixed endothelial cells and the large mononuclear leucocyte. They seem readily to engulf particles which arrive on their surfaces, and to proliferate under the stimulus, become detached, and either to be cast off in the expectoration or taken up and carried along the course of the lymphatic absorption. The cells of the upper respiratory membrane, on the other hand, do not appear to be phagocytic, but rather to throw off foreign particles by means of their secretion.

Rabbit 3 was given one injection of india ink and killed twenty-four hours later. Autopsy showed traces of ink along the respiratory tract, with massive blackening of the upper lobe of the left lung and moderate spotting of the middle lobe. The rest of the

lungs appeared practically free from carbon. The bronchial nodes were moderately discolored. The cervical and mesenteric nodes were negative. Microscopic sections of the lungs gave similar findings to those in rabbits 1 and 2. Sections of the spleen and kidney showed slight traces of carbon.

Rabbit 4 was given about three cubic centimeters of india ink at one time and killed half an hour later. Autopsy showed much ink in the nose, mouth, pharynx, larynx, esophagus, trachea and bronchi. The stomach was filled with vegetable material, and this was blackened over a radius of about two centimeters around the esophageal opening, but there was no discoloration below this sharply defined level in the digestive tract, the remainder, even of the stomach contents, being free from ink. The lungs showed almost complete blackening of the left upper lobe and marked blackening of the left lower and right middle lobes. The bronchial nodes showed marked hyperemia and a moderate amount of carbon scattered through them. The cervical and mesenteric nodes were negative. The presence of carbon all along the inhalation route, with its complete absence in the intestine, completes the proof that the lung findings are due to inhalation.

Rabbit 5 was given 0.5 cc. of an emulsion of human tubercle bacilli in the nose. In this case there was not the slightest, even momentary, disturbance of the respiration. It was killed and examined seven weeks later, when it showed no lesions of the respiratory mucous membrane above the lungs. The lungs showed advanced tuberculosis of the right upper and middle lobes with consolidation, and moderately advanced tuberculosis of the bronchial nodes. The cervical nodes, spleen and mesenteric nodes were all negative both grossly and microscopically.

Rabbit 6 was given 0.75 cc. of an emulsion of tubercle bacilli and killed after seven weeks. The autopsy findings, gross and microscopic, were identical with those in rabbit 5, the tuberculous process being limited to massive infiltration of the upper and middle lobes of the right lung and moderately advanced lesions of the bronchial nodes.

Where the time conditions permitted, the injection material was found all along the respiratory tract to the terminal bronchi

and the alveoli. There was never evidence of lesions of the digestive or upper respiratory tract nor of absorption from them by the mesenteric or cervical lymphatics. The lung findings were no less striking when the animal was killed before the injection had gone further in the digestive tract than the cardiac end of the stomach. Comparatively little of the injected materials penetrated the circulation beyond the lungs and their lymph nodes; not enough in the case of the tubercle bacilli to cause any lesions elsewhere in seven weeks. In five of the six animals the maximum effect was produced in one upper (apical) lobe, in the sixth case in both upper lobes, while in only two cases was there an equivalent effect in a middle lobe and in only one case in a lower lobe."

The fact of most importance in these experiments is that the bronchial nodes were involved in every instance. In lymphatic absorption from the sinuses, the deep cervical chain of lymphatics were involved, as well as the bronchial nodes. In the inhalation experiments only the bronchial nodes were involved.

Of interest in connection with these experiments is the report of Quinn and Meyer,³ who reviewed all the literature on the subject, and quoted Dunham and Skavlen⁴ as saying that 73 per cent of their patients with bronchiectasis had infection in the upper air passages. Quinn and Meyer found the two conditions associated in almost 58 per cent of their own cases. They showed also that while the patient is asleep, the aspiration of iodized oil into the lung from the nasal fossæ is possible and does occur with apparent ease, and that not infrequently the amount aspirated is surprisingly large. They felt that this was probably the most important factor in explaining the frequent concomitant occurrence of bronchiectasis and sinusitis. McLaurin⁵ of Dallas also has found lipiodol in the chest after placing it in the antrums.

William Ewart⁶ has made the following statement in Albutt and Rolleston's *System of Medicine*: "The terminal bronchial tubes are vascularized by the pulmonary artery. The peribronchial lymphoid masses are said to occur on the side of the bronchus opposite to that occupied by the accompanying pulmonary artery. The activity of the alveolar lymphatics is shown by the rapid absorption of the products of pneumonia." From the same

source comes this statement regarding the cause of bronchial dilatation: "A fourth group is that in which a temporary or permanent narrowing of a large bronchus, as by an aneurysm, has led to increased strain or to accumulations within its subdivisions." Dr. Achard was quoted as having found that the cause of bronchiectasis, verified by necropsy, was chronic bronchitis (e. g., cough, onset gradual) in 45 per cent of cases, and that a history of chronic cough since childhood was present in 15 per cent of the cases.

In 1927, Clerf⁷ made a study and recognized the association between chronic sinus infection and secondary infection in the chest, and it was his opinion that both the inhalation and lymphatic routes that I had described might function in producing chest infections.

Childrey and Essex⁸ referred to the experiments by Dr. Ryder and myself, but from the results of their own work they concluded that the "mucosa of the sinuses is highly resistant to absorption." In the studies they made, observations were made upon normal sinus mucosa; in a few the sinus was infected, but the animal was killed very soon after the dye was injected.

Some of our experimental animals were kept alive thirty-seven days before autopsy was done. It would seem that the thickened edematous mucous lining of the sinuses, often infected and sometimes containing encapsulated abscesses, is capable of giving slow absorption.

Let me quote from our experiments:¹ "If the point of the needle was free in the cavity of the antrum, some of the ink nearly always escaped into the nose and ran out at the nostril. However, we found that ink was quite readily carried away from the nasal fossæ by secretion, without being absorbed, while it stagnated in the antrum and was in part taken up by the lymphatics. In one rabbit, the point of the needle, after passing through the left antrum, penetrated the mucosa of the inner wall, and about one cubic centimeter of ink was injected beneath the mucosa."

Baum⁹ has referred to the theory of lymphatic drainage from the sinuses as one of the obstacles that had to be overcome to establish his point that sinus disease is not a cause of bronchial

asthma, and in his statistics he classified together asthma, bronchitis and bronchiectasis. This I feel is wrong, since asthma and bronchiectasis are in no way comparable, and whatever views one may hold as to the influence of infected sinuses upon bronchial asthma have no place here. However, granting in either case that the consideration is theoretical, certainly the manner of the production of these two chest conditions must differ entirely.

Lanson¹⁰ has said that it is only in the complicated, chronic sinus infections with edema, polypoid degeneration and putrefaction that noticeable systemic symptoms are produced. He feels that there may be edematous changes similar to nasal polyps, the connective tissue may be thickened and retention cysts may be caused by round cell infiltration constricting the necks of the glands. According to Lansen, "Toxemia in chronic disease of sinus membrane is of great clinical significance and probably originates from the absorption of small quantities of highly virulent toxins generated within the sinus cavity. Whether the important amount of absorption occurs through the sinus membrane or direct from more highly absorptive tissues which receive the toxic material from sinus drainage is a subject for observation. Clinical observation would seem to bring out the fact that secondary infections are usually blood-borne."

I have discussed this subject frequently,¹¹ and have very little reason to change my theories concerning the routes of infection from the sinuses to the chest. My only motive in reviewing the subject is that my views apparently have been misunderstood by many rhinologists and medical men. Sinuses have been operated upon in a large number of cases in which they should not have been touched. Confusing glands in the chest, which I think are infected from the sinuses, with those in which the infection is due to tuberculosis, and also failing to grasp the fact that a child with peribronchial tuberculous glands might coincidentally have infected sinuses, have been sources of very serious errors made by physicians. These errors are only exceeded by operating upon the sinuses of the child who has puerile tuberculosis of the chest.

I should like to repeat some of my statements regarding this problem which were published in 1920:¹² "Sure as I feel of the causal relationship between sinus and bronchi in these cases, I do

not feel that we are in a position to be too dogmatic as to exactly how the process develops. As I have said, we have demonstrated the lymphatic route from sinus to lung, and I feel sure that it often functions in the development of the bronchial condition as a secondary lesion from the primary antrum. In cases where there is little or no escape of pus from the antrum into the nose, I think the lymphatic route is probably the path of infection par excellence. On the other hand, in cases where the nose is constantly full of pus, it is possible that inhalation may be more important than absorption. Some experiments now in progress and not yet reported illustrate strikingly the readiness with which substances constantly present in the upper air passages reach the lungs by inhalation and we cannot wisely ignore this fact in trying to interpret the relation between infections of the upper and lower respiratory apparatus. Moreover, other regions than the accessory sinuses may well be the source of lymphatic infection of the lung."

"Against this interpretation has been urged the fact that cure of the diseased sinus does not always result in cure of the diseased lung. But neither does cure of an infection of the foot always result in cure of a phlebitis or adenitis secondary to it. It depends on how far the secondary lesion has progressed, and the same is true of our sinus-bronchi cases. If the bronchial infection is in an incipient state, cure of the sinus, thereby removing the source of bronchial infection, will often result in complete recovery. If, on the other hand, the bronchial disease has become well established—a severe chronic bronchitis, with or without bronchiectasis—treatment of the sinus, however successful, is not likely to relieve this. It must be treated directly by postural drainage, autogenous vaccine, etc. In cases of the intermediate type with acute exacerbations, the attacks can be cut short and chronic bronchitis averted in many cases by treatment directed to the sinuses."

Whenever chest conditions are discussed, all authorities quote the immortal Laennec. It seems to me appropriate in the present discussion to interject some interesting and rather pertinent facts from Laennec's own personal history. I quote freely from a memoir written by my friend, Gerald Webb:¹² "Since his [Laen-

nec's] residence in Paris, he had suffered with what he termed asthma; to combat it he turned to bizarre remedies. One day, in an effort to continue at his studies he sat at his desk with his body naked. . . . Laennec attributed bronchiectasis to the effects of any malady which would cause coughing in a prolonged and violent way over a considerable period of time, especially a disease like whooping cough. . . .

"In April of 1826 he contracted a severe cold, but continued at work. A throat condition followed, accompanied by high fever and pains in the chest. In May he commenced to cough, with expectoration being markedly fetid. . . . An affected upper molar tooth was discovered and extracted; a great deal of fetid pus was discharged, probably from the antrum. It is the conjecture of Rouxneau that all symptoms, including the cough and diarrhea, were due to this infection. . . . The active fever continued, the expectoration was very profuse, and pus still was discharged from the maxillary sinus. . . . Laennec's cousin, Ambroise, came from Nantes to make an examination. This physician found bronchophonic resonance, the respiration vesicular, mucous and sibilant râles at the base of the left lung."

Laennec died the 13th of August, 1826; no autopsy was performed. However, I believe from this report that Laennec had bronchiectasis and sinus infection.

SUMMARY.

To recapitulate, I believe that protracted flooding of the lymph channels with infectious material from the hyperplastic maxillary sinus tends to produce a chronic peribronchitis. This is often clearly shown in the roentgenograms of children. Frequent chest colds may be the first evidence of this extension, but a definite chronic bronchitis is eventually established and in a neglected case the final result is a bilateral bronchiectasis, which, when well established, is, of course, permanent.

A sharp distinction must be drawn between those cases of bronchiectasis which result from chronic sinusitis and those which are due to other causes. Thus, besides infections of the paranasal sinuses, the common etiologic factors of chronic bronchitis and

bronchiectasis are: lung abscess, foreign bodies and fibroid phthisis. In fact, any long continued suppuration in the lung tends to produce deformity and dilatation of the bronchi; in such cases the infrequent presence of a sinus infection is only a coincidence. It is usually possible to identify the cause of the chronic lung suppuration in these cases, and they, moreover, usually show definite anatomic characteristics which differentiate them from those cases in which sinus infection is the exciting cause.

The association of sinus disease with bronchiectasis is far too common to be accidental. The most typical cases start in childhood. A careful history usually gives the information that the child has been subject to frequent and protracted colds, with great tendency to cough. The onset of the sinusitis may be traced to some one of the acute infectious diseases, measles and whooping cough being notorious offenders. A chronic cough with bronchitis follows. The child is usually undernourished and may or may not have symptoms of an involvement of the nose or sinuses. Freedom from symptoms, therefore, should not prevent a careful clinical and roentgenologic examination of the sinuses. At the same time stereoscopic roentgenograms of the chest, in the anteroposterior and the oblique positions, will show some degree of peribronchial enlargement and a thickening of the bronchi at the bases. If this condition is not corrected, the eventual course may be pansinusitis with bilateral diffuse bronchiectasis.

CLEVELAND CLINIC.

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LXI.

ANAEROBIC RETROPHARYNGEAL ABSCESS.*

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Retropharyngeal abscess is well understood and has been frequently described. It occurs in infancy and early childhood in a great majority of cases. This type of suppuration originates in one side of the posterior pharyngeal region and indicates the supuration of an inflamed paravertebral lymph node. It usually remains localized to the side of its origin. Occasionally we encounter a unilateral abscess in an adult which is the result of the ultimate localization of a peritonsillar infection.

The anatomy of the postpharyngeal structures is well known to the laryngologist. There is very little distance between the mucous membrane and the vertebral column under normal conditions. This distance is occupied by pharyngeal fascia, thin muscle which consists of the three constrictors of the pharynx and the longus colli of either side. These are enveloped in fascia and are separated from the vertebral bodies and their fascial covering by loose areolar tissue.

The anaerobic abscess differs from the usual type of abscess in this region principally because the localized infection is caused by one or more anaerobic organisms and because it is caused by a foreign body, usually a bone, which inoculates the retropharyngeal space with anaerobic bacteria. If the lodgment of the bone is on either side the lesion will commence as a unilateral one. If the process is allowed to progress before it is incised this type of abscess appears to become central. The median raphe of the constrictor muscles probably influences the lateral localization in these cases as it does in the usual type of abscess.

The local process shows the same effects as are seen from anaerobic infections elsewhere, with the exception that the offend-

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ing organism or organisms do not appear to be as destructive and as dangerous to life as such organisms as the Welch bacillus which causes true gas gangrene. It is, of course, possible to have an infection with such an organism. In such a case the same rapid extension with necrosis and serious toxemia would supervene as occurs with similar infections elsewhere in the body. Recently Hunt¹ had a case with such an infection. His patient succumbed a few hours after she had swallowed a piece of chicken bone. It has been difficult to learn much from the literature on anaerobes which might apply to the subject under discussion. There appears to be much confusion in bacteriologic literature concerning these organisms despite the brilliant efforts of numerous workers.

However, both cases which are reported in this paper showed the following characteristics of this specific type of infection. There was a great amount of slough, due to the proteolytic action of the organisms, and roentgen study revealed the presence of air and fluid in the retropharyngeal space. One of the abscesses was centrally placed, and both lasted much longer than the usual type of abscess. Both presented slight external swelling of each side of the neck along the sternomastoid region.

The etiologic factor in each of the cases was the penetration of a bone or spicule of bone beyond the surface of the mucous membrane.

The clinical histories follow :

Case 1.—N. M., female, age 59, edentulous, swallowed a spicule of chicken bone, which stuck in the back of her throat, two days before admission. She was sent to the hospital by her physician because of a slight swelling of the neck. Examination revealed a moderate amount of edema about the angle of the jaw on each side, extending downward along the neck and forward toward the floor of the mouth. Pressure upon the front of the neck elicited pain. At this time her throat presented a most unusual picture. (Fig. 1.) There was a moderate degree of retropharyngeal bulge. But the most significant thing was a peculiar, glossy, jelly-like appearance of the pharyngeal wall which was more pronounced in the lateral folds. (See Fig. 1.) Palpation of these structures elicited the same type of crepitation that one finds in



Fig. 1 (Case 1). Picture at the time of admission two days after the ingestion of a spicule of bone. No pus obtained on incision. Crepitation of lateral folds on palpation.



Fig. 2 (Case 1). Four days after admission, six days after trauma. Note fluid level with numerous air spaces intermingled with pus. Incision yielded foul gas and pus.

subcutaneous emphysema. By means of the direct method of examination with the patient recumbent, what was probably a very small spicule of bone was dislodged from the posterior pharyngeal wall at the level of the base of the tongue. There was an intense edema and some swelling extending downward as far as the cricopharyngeus. The esophagus appeared normal. Two incisions were made into the bulging tissues, as a result of which a small amount of thin serous fluid was evacuated. There was no odor. As a result of the incisions and multiple punctures the patient felt somewhat better. The gas in the lateral folds had disappeared the next day. The patient was closely watched. Three days later a retropharyngeal swelling which filled most of the pharyngeal space and was beginning to encroach upon the larynx was to be seen. (See Fig. 2.) As the swelling progressed it became soft and doughy, presenting a peculiar resistance to the palpating finger. Incision at this time yielded a large amount of very foul smelling, brownish, thin fluid which was accompanied by an escape of gas having the same extremely foul odor. The odor was the same as is encountered in putrid lung abscess. From this time on the wound was kept open and the cavity was swabbed with 50 per cent peroxid of hydrogen solution. Of interest is the fact that considerable slough began to discharge through the wound on the fifth day after evacuation of the abscess. The cavity gradually became smaller until it was declared healed on the thirty-fifth day after her illness began, when she was discharged. (See Figs. 3, 4 and 5.) Bacteriologic studies revealed a pure culture of a gram negative anaerobic bacillus.

Case 2.—F. C., female, age 45, edentulous, came into the hospital with a fishbone buried in the retropharyngeal tissues. (See Fig. 6.) She had swallowed the bone twenty-four hours before admission. As a result, she developed pain in the right side of her throat which gradually became worse. There was a slight swelling of each side of the neck, and tenderness was elicited by pressing the larynx backward. (See Figs. 6 and 7.) Shortly after admission, by means of the direct method, Dr. Kramer found a bone protruding into the right hypopharynx, which he removed. Drainage through the fistulous opening, left by removal of the bone, was insufficient, so that on the sixth day the



Fig. 3 (Case 1). Four days after incision. At this time there was slough formation.



Fig. 4 (Case 1). Seventeen days after incision. Only slight amount of exudate but still some slough.



Fig. 5 (Case 1). Lipiodol used to demonstrate the remains of the abscess space. No air can be demonstrated. About ten drops of pus per day at this stage. Completely healed five days later.

abscess was incised and foul pus was obtained. X-ray study revealed the bone in the retropharyngeal wall and space surrounded by numerous gas bubbles. The abscess area was watched and treated daily. It was completely evacuated and the wound healed at the end of fourteen days. Anaerobic studies were not carried out in this case. *Streptococcus viridans* and *staphylococcus albus* were present in the exudate. The pus was white in color and a small amount of slough was discharged from the wound.

SYMPTOMATOLOGY.

1. Pain.—Pain is present while the bone is in situ. After the bone is removed there is very little spontaneous pain, such as is encountered in subcutaneous anaerobic abscess, because the pre-vertebral pharyngeal tissues are loosely applied to the underlying structures and will therefore permit forward displacement with very little resistance.

2. There is difficulty in swallowing and some pain during swallowing because of the bulging mass and because of the tension upon the constrictor muscles of the pharynx.

3. The toxemia was not severe in either case.

In case two, the temperature rose as high as 104 immediately after the extraction of the foreign body; otherwise the temperature range was below 101.6.

Differential diagnosis should be made.

1. From simple retropharyngeal abscess which is unilateral and occurs in infants in a great majority of cases.

2. Edema and swelling due to extension of deep seated supuration, such as a peritonsillar abscess burrowing posteriorly. This is unilateral and the antecedent history is helpful.

3. Caries of a vertebral body with cold abscess—the duration, chronicity and X-ray help differentiate this condition.

4. A similar unilateral abscess could be caused by inoculation of infection with a tonsillectomy needle.

Diagnosis.—The diagnosis of retropharyngeal abscess of the anaerobic type is readily made. The following features of this condition are worthy of consideration in the matter of diagnosis:



Fig. 6 (Case 2.) Showing bone penetrating retropharyngeal tissues at the level of the crest of arytenoid. Air is present in the tissues. Picture taken twenty-four hours after ingestion of bone.



Fig. 7 (Case 2). Showing evidence of gas and pus formation in the retropharyngeal space.

1. A history of injury to the postpharyngeal region by a bone.
2. A posterior pharyngeal bulge, either centrally or laterally placed.
3. X-ray shows an opaque shadow with a fluid level and peculiar mottling which indicates the presence of gas.
4. Crepitation of the local tissues, if seen early enough, or at the opportune time to observe this phenomenon before pus formation occurs.

5. Fluctuation when fluid has accumulated. There is a peculiar sensation of resistance. This is no doubt due to the admixture of gas with the fluid.

6. Dysphagia and odynophagia.

Dyspnea may supervene if the abscess is allowed to progress.

Symptoms such as spasm of the glottis, change in the voice due to collateral edema, cough and nasal regurgitation of fluids could occur in this type of abscess as they do in the other.

The positive diagnostic procedures are as follows:

1. X-ray which shows the presence of gas.
2. Aspiration with a needle or exploratory puncture with a fine ear knife.

Both these procedures are safe and will demonstrate the presence of foul gas and foul exudate without permitting a flooding of the pharynx and possible aspiration.

Treatment.—Careful and adequate surgical treatment of these cases is important. Because of the anaerobic organisms which are known to cause such serious disease in the lung, we must be extremely careful that none of the abscess content reaches the tracheobronchial tree. The writer has always used the technic which follows in retropharyngeal abscess.

With the aid of any suitable direct instrument, such as the Mosher or Jackson laryngoscope or the short laryngeal tube of Yankauer, a fine pointed knife is forced into the abscess cavity. Its withdrawal is accompanied by a trickle of pus. A laryngeal suction tube is now placed through this opening and the contents of the cavity are completely aspirated. The opening is then enlarged by a longer incision with a larger knife, and this incision is stretched so that a gaping opening into an empty space results. The first case which was treated by the writer was cared for in

the above manner. In addition, daily attention to the abscess area consisted in maintaining the patency of the opening and swabbing with 50 per cent peroxid of hydrogen solution.

The local process should be frequently checked by means of X-ray studies. The treatment may be summarized as follows:

1. Remove the foreign body.
2. Evacuate the abscess cavity.
3. Keep the opening to the cavity patent.
4. Introduce oxygen in some form into the abscess cavity.

Peroxid of hydrogen seems to be effectual. Oxygen gas could be introduced directly into the cavity by means of a cannula but should not be under pressure.

Complications.—Because of the sloughing and the longer duration of these abscesses it is reasonable to expect that complications, such as thrombophlebitis of the internal jugular vein with systemic invasion by bacteria could occur. If untreated such abscesses might cause laryngeal stenosis, due to edema, and might rupture spontaneously, causing asphyxia or lung abscess. Lung abscess might readily follow if the respiratory passages were not protected during evacuation of the abscess.

Considerations.—For much of what follows I am indebted to the excellent monograph which comprises the reports of the Committee upon Anaerobic Bacteria and Infections, of Great Britain.²

One is impressed with the similarity of the local process in anaerobic retropharyngeal abscess and that in simple subcutaneous abscess due to anaerobic infection. The period of time which elapses before a wound shows signs of infection varies. This may occur within an hour, or several days may elapse. The brownish color is thought to be due to the breaking down of blood clots. In case two, the color of the exudate was whitish. This might be accounted for by the type of anaerobic organisms or by the presence of staphylococcus albus which was isolated from cultures of the wound. The odor of the exudate appears to depend upon the type of offending organism and upon the stage of infection. There is no odor present in the early stages. There is a local swelling, due to the accumulation of serous fluid in the subcutaneous tissues. This is in accordance with our experience in case one. In speaking of localized infection, the

report states: "Throughout the precincts of the wound there is an infiltration of gas, and it extends to further limits along certain lines, the subcutaneous tissues, the perivascular tissues and the planes of intermuscular connective tissue. Up to this point in the clinical history of the wound the muscle is not infected. When it does become infected the nomenclature must be altered. It is no longer an anaerobic infection of the wound but the condition of gas gangrene." Because of the anatomic arrangement of the tissues in the retropharyngeal region there is less likelihood of the production of a condition of gas gangrene.

Bacteriology.—As early as 1776, Spallanzani³ mentioned organisms that caused fermentation, and in 1861 Pasteur⁴ carried out studies upon organisms which lived without oxygen and generated gas. The following statement of the research committee is of significance: "The minor or comparatively unimportant type of anaerobic infection known as gas abscess in which simple incision and free drainage are generally sufficient treatment, corresponds no doubt to the results obtained in animals when whole cultures of nonpathogenic or washed pathogenic anaerobes are injected into the subcutaneous tissues (except that in the wounded man there is generally a sufficient quantity of necrotic muscle to provide a fermentable pabulum.)"

And further, "The rôle played by the more numerous but less dangerous nonpathogenic anaerobes has not been clearly defined; they may be responsible for some of the features of gangrenous wounds, such as putrefactive odor, blackening and digestion of the tissues. It should be noted that any one or a combination of these organisms may be found in a wound which is not and does not become gangrenous."

SUMMARY.

Two cases of anaerobic retropharyngeal abscess are recorded. This condition is a different clinical entity than the retropharyngeal abscess which is usually encountered. It is caused by the ingestion of a fish or chicken bone and is characterized by the presence of a sloughing, foul odor and gas. It lasts longer. It is located according to the site of penetration of the bone. This

form of abscess is well illustrated by the roentgen film, and is apparently not dangerous to life. In the first case an anaerobic gram negative bacillus was isolated. This could not be identified. Streptococcus hemolyticus was also isolated from the wound. Anaerobic studies were not carried out in the second case, but cultures for aerobes disclosed the presence of streptococcus viridans and staphylococcus alba.

The bacteriology of this condition, which embraces a phase of anaerobic bacteriology, is poorly understood. Mention should be made of the cases of perforation of the esophagus⁵ and para-esophageal abscess with foul exudate, which is evacuated at the time of incision. A suggestion is offered that such esophageal cases be studied closely with the X-ray.

12 EAST 86TH ST.

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LXII.

ANATOMIC PHASES INVOLVED IN THE SURGERY
OF THE NASO-ANTRAL WALL AND FLOOR
OF THE MOUTH.*

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The points which occupy our attention during surgical procedure remain anatomic long enough to satisfy hemostasis and to protect an irrelevant vital function. We are mainly anxious to afford surgical drainage to an acute or chronic infection and remove diseased tissue. Fortunately we have surgical license to disturb or destroy adjacent structure, if the best interest of the tissue demands it. Even as simple a thing as the ostium of the nasolacrimal duct, protected as it is by its own construction, happens to occur in the naso-antral wall, the site of punctures and resections, the most frequent surgical efforts in otolaryngology, except tonsillectomy. It seems well, therefore, to briefly review normal variations of this structure and also to mention the important source of blood supply in the posterior edge of the naso-antral wall.

The nasolacrimal canal, formed and protected by the bone of the maxilla, the lacrimal bone, and the lower turbinate, empties into the lower nasal meatus, by its ostium. This ostium occurs just below the attached border of the lower turbinate, on the lateral wall of the lower nasal meatus, usually very highly placed, and about 30 to 40 mm. from the anterior nares. It is shown by Schaeffer to appear at various levels almost to the floor of the nose, but to keep its fairly constant distance from the anterior nares. The nasolacrimal ostium may be single or multiple, and varies normally from a large, flat, spoonlike opening, to a dot surmounting a small nipple. It is sometimes a small slit, which

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is merely a potential opening. These types Schaeffer¹ has classed as about eleven, some of which are reproduced here. Ten of these types are in the upper fourth of the lateral wall of the lower meatus, and only one close to the floor.

We have the assurance of investigators of long experience that harm rarely comes to the nasolacrimal ostium in nasal surgery, on account of its protected position and its open, gutterlike shape. However, in the daily run of patients the complaint of tearing after an operation, weeks or years before, is regular and impressive. Analyzing this, it would seem that the source of traumatic

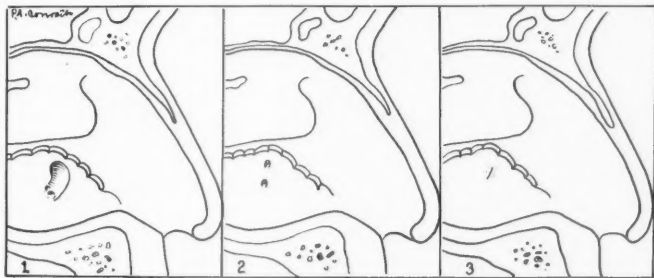


Fig. 1. Types of ostia of the nasolacrimal duct. (Slightly modified after Schaeffer, "Nose and Olfactory Organ," p. 251.) No. 1 is the wide mouth type most seen, shown in most frequent location. No. 2 is a type of ostium with multiple opening. No. 3 shows a common ostium structure, of slit-like arrangement. Wide variations occur in distance from nares, distance to floor of nose, and in size. Type 1 is most frequent, located within upper fourth and anterior third of suborbital naso-antral wall.

eschar comes from injury to the ostium when it is low or within site of the antrum window.

Not so much can happen to the larger ostium. Only in a most vigorous effort to bring an antrum opening forward to the anterior angle, or a deep section of the anterior attachment of the lower turbinate, can a large ostium be harmed. With the smaller ostia, scarring is easy, and blockage is often made by hypertrophy of the lower turbinate, or edema. Much of the tearing incidental to hay fever is due to the edema and hypertrophy of the lower turbinate.

Correction of stenosis of the nasolacrimal duct has been the subject of study for many years. The surgical treatment of maxillary

sinus disease by resection of the naso-antral wall is so frequent that it becomes a real factor in the cause of stenosis of the lacrimal duct. The location of its ostium should be constantly borne in mind.

In making the naso-antral window it was my custom for a time to take the posterior limit of the wall back as far as possible, using a Jansen-Middleton forcep through the Luc-Caldwell opening, or the lower meatus, of the nose. One shocking hemorrhage, in a case which finally required ligation of the external carotid,

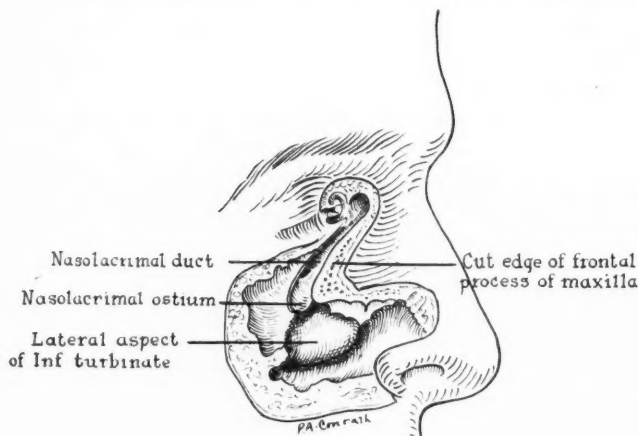


Fig. 2. Sketch of dissection made after Schaeffer, "Nose and Olfactory Organ," p. 245, to show relation of average ostium of nasolacrimal duct to inferior meatus, naso-antral wall, and lower turbinate.

inspired me to ever after resect the posterior part of the window with caution. Frequent reference is made to the distribution of the posterior lateral nasal arteries, four in number, all branches of the sphenopalatine artery. Clearest of these descriptions is by Schaeffer,² and recently in a clinical anatomic thesis by Neivert.³ The lowest branch is small, runs close to the nasal floor and supplies the nasal wall with branches upward. One of the two larger branches comes off at the point where the lower posterior turbinate tip is attached. The case (Gerloch, age 28), mentioned above, made me wonder if I had sectioned the artery

in the wall with the forcep, or had torn it, when the turbinate was infracted upward.

Now I wish to refer to certain cases of peritonsillar infection involving the floor of the mouth. When these involve the floor of the mouth and extend to the pharynx, they have been known as Ludwig's⁴ angina since his description in 1836. He noticed that most of the cases died of suffocation from crowding of the pharynx and larynx with septic edema of the tissues. In the nu-

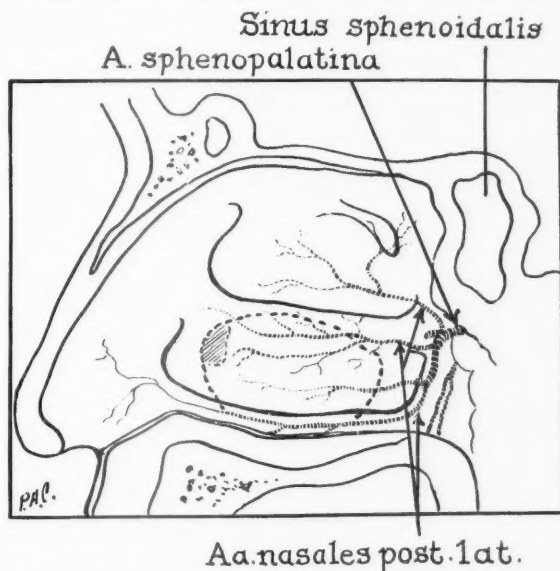


Fig. 3. Drawing of a dissection of the lateral wall of the nose, for arteries. Shaded area shows average site of ostium of the nasolacrimal duct. Dotted line indicates average removal of naso-antral wall at operation.

merous case reports which flooded the literature in the following fifty years, more attention was paid to study of the cause and treatment than to the anatomic structure controlling course of the disease. Frequent mention was made of the rapidity with which infection passed to the pharynx and larynx region. Poulsen tried to show the route by lime injection. He stated that it reached the larynx by way of the retromaxillary fossa, through lymphatics, thence through the wall of the pharynx.

Delorme, Huguet and DeBovis,⁵ in 1894, and others considered the infection a "sublingual phlegmon" of muscular origin, crowding the larynx by swelling of the tongue. They based their proof on the fact that an external incision capable of relieving the pus had to pass through the mylohyoid muscle. Nelaton, Chauvel and others describe sublingual incisions in the floor of the mouth, most of which failed of their object. The records of Semon,⁶ an important study of a large experience, detailed the clinical process, and, like the others, stress the speed with which the mass infection moved to the pharynx. The high mortality to this time showed that the efforts lacked a perfect understanding of the anatomy. Thomas,⁷ in 1908, in a classical study of 106 cases, defined the route taken by the infection, as follows:

"The muscular floor of the mouth is formed by the two mylohyoid muscles, which fuse with each other at the anterior median raphe. This muscular diaphragm separating the mouth from the neck is a complete one from the posterior edge of one mylohyoid muscle to that of the other and is a comparatively strong one. There are no openings in it for the passage of planes of connective tissue between the mouth and neck. From the posterior border of the mylohyoid on each side extend backward the constrictor muscles of the pharynx, separating the pharynx from the neck, the muscles of the two sides fusing together at the posterior median raphe. The three constrictors, superior, middle and inferior, overlap each other, so that here, also, the submucous tissue of the pharynx is not continuous with the connective tissue of the neck through these muscles. Between the posterior edge of the mylohyoid and the anterior border of the middle constrictor, however, is a considerable deficiency in the buccopharyngeal muscular wall. . . . Those structures which pass from the neck into the mouth or in the opposite direction do so through this opening. These are the glossopharyngeal and hypoglossal nerves, the lingual artery and vein, and the styloglossus muscle. The greater part of the opening, however, is occupied by the deeper portion of the submaxillary salivary gland, which here projects into the floor of the mouth, near the root of the tongue, where it lies just under the mucous membrane."

Numerous researches have been made as to the etiology and character of Ludwig's infection, but the above clear description of the route taken cannot be improved. Dissection of the floor of the mouth at this point requires an incision through the mucosa, a blunt spreading of connective tissue mesial to the deep part of the submaxillary gland, and the forcep passes with ease directly backward and mesially into the parapharyngeal region. Certain of the simpler cases of cellulitis of the floor of the mouth after

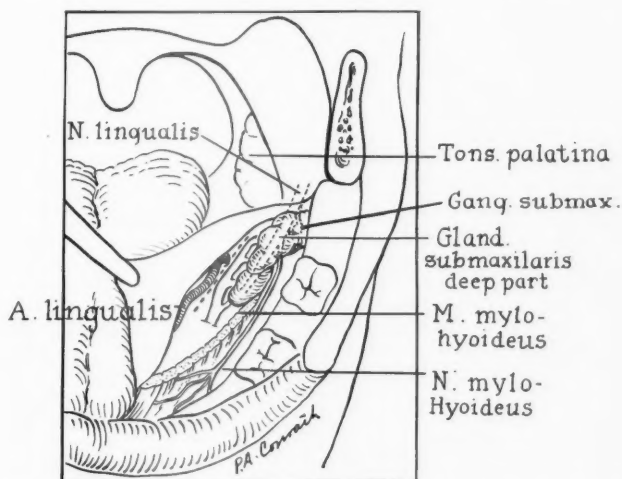


Fig. 4. Drawing of a dissection of the floor of the mouth, showing principal structures involved in blunt dissection toward parapharyngeal space. The unlabeled dotted line shows about where mucosa incision is made.

molar infection can be drained in this way if vigorously localized before incision.

722 BEAUMONT MEDICAL BUILDING.

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LXIII.

THE LARYNGOLOGIC ASPECT OF HODGKIN'S DISEASE: REPORT OF A CASE.*

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The laryngologist is repeatedly confronted with the problem of making a differential diagnosis in cases presenting enlargement of the cervical lymph nodes. The majority are simple cervical adenitis and subside with the healing of the acute infection, but occasionally the nodes remain swollen. Every patient presenting enlargement for over a month of the cervical lymph nodes should be advised to watch these apparently simple glands.

The following case is of interest because the pathologic picture of lymphatic tuberculosis and lymph granuloma (Hodgkin's disease) were found in different nodes removed for biopsy.

E. C., age 6, female, was referred on April 22, 1929, by Dr. E. C. Mitchell, for the removal of a growth behind the right tonsil fossa, with a report that a physical examination of the lungs was negative.

Family history: Two maternal grandaunts died of tuberculosis. The child's past history was negative, save for an occasional sore throat. One year previous to admission to the hospital, her tonsils and adenoid had been removed, as a possible cause of the enlargement of the right cervical glands. In spite of tonsillectomy, the glands increased in size and the condition spread to the remaining cervical glands of the same side. One of the older glands was removed from this side and reported as being diagnostic of Hodgkin's disease (Memphis Eye, Ear, Nose and Throat Hospital).

The general physical examination of the child was that of a well nourished and developed girl for her age. There was a uniform enlargement of the lymph nodes of the right side of the

*Presented before the American Laryngological Association, Atlantic City, May, 1932.

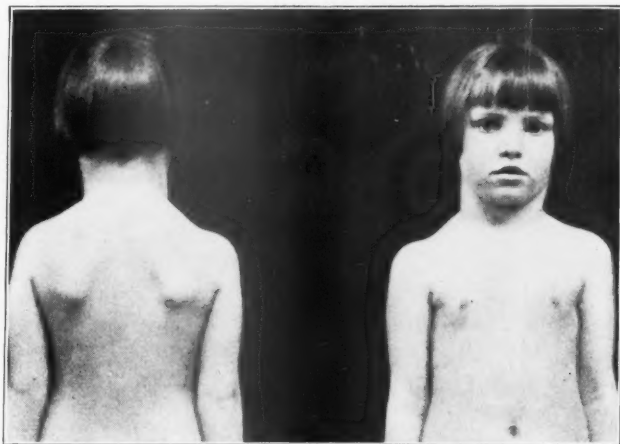


Fig. 1.

Taken April 22, 1929, showing bilateral enlargement of cervical glands.

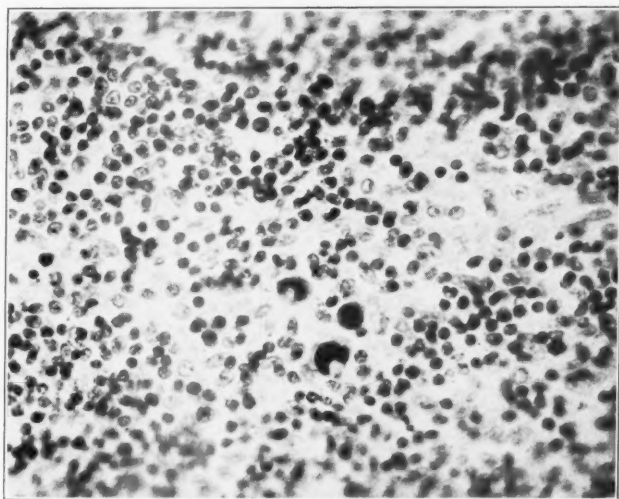


Fig. 2. Section of right cervical lymph gland removed two years after onset. Showing diffuse hyperplasia of lymphocytes and endothelial cells with mitotic figures, large numbers of eosinophile cells and frequently a Dorothy Reed giant cell. Diagnosis: Hodgkin's disease.

neck and a lesser enlargement of those of the left side. A recent skin incision of the right side of the neck was healing.

The individual cervical nodes were discrete, the largest being one inch in diameter and anterior to the sternomastoid muscle. No other glands were palpable. Von Pirquet and intradermal tuberculin tests (1 mg.) were negative.

The tonsils had been thoroughly removed, but the right half of the pharyngeal wall was displaced forward by a mass, which was solid. The mucous membrane covering the mass glistened but lacked any of the signs of an acute inflammation. The lingual tonsils were normal. No pathology was visible in the naris.

Operation: Under ether anesthesia an incision was made through the membranous layer of the pharyngeal wall, exposing the nodes. The growth was readily dissected free, as there were no fast adhesions, and the mass was removed with a tonsil snare. The cavity was closed with interrupted sutures. An uneventful convalescence ensued and the pathologic diagnosis was chronic hyperplastic tuberculosis.

A month later, the right chain of the glands was removed for study, and the diagnosis was chronic hyperplastic tuberculosis. Eleven months later, the left cervical chain having enlarged, was removed and the diagnosis was Hodgkin's disease. Again in six months a second gland was removed from the left side with a diagnosis of Hodgkin's disease. A year later, I saw the girl after an interne had failed to open what he thought was a left pharyngeal abscess. The right half of the pharynx was flat, but the left pharyngeal chain was now enlarged. The child has maintained a good state of nourishment and is growing. The enlargement of the glands in the right side of the pharynx is half of that of the left, and both are moderately firm, with no areas of fluctuation or tenderness.

BACTERIOLOGIC FINDINGS.

A portion of a right cervical lymph gland was removed in a sterile container. This was macerated in a sterile agar mortar and ground up with sterile broth. One c.c. of the finely divided suspension of this material was injected into the groin of a guinea pig, whose greatest susceptibility is to the human strain of the

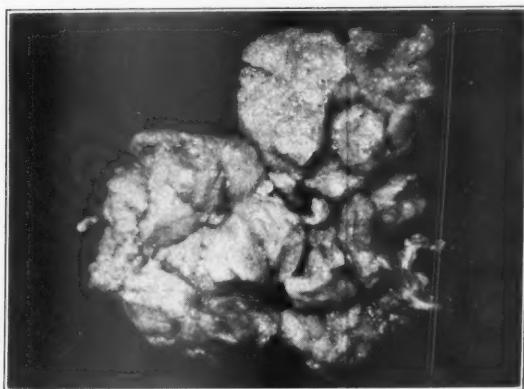


Fig. 3. Showing a somewhat torn, greatly enlarged lymph node from the right posterior pharyngeal wall, which was removed twenty-two days after the right cervical gland.

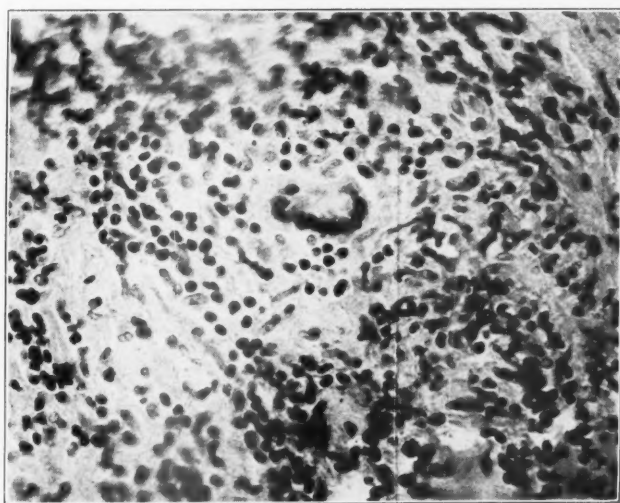


Fig. 4. Section of the gland from the right posterior wall, showing epithelioid and lymphoid cells about a tuberculous giant cell forming an early tubercle. Diagnosis: Chronic hyperplastic tuberculosis.

tubercle bacilli: $1\frac{1}{2}$ c.c. into the peritoneum of a rabbit, whose greatest susceptibility is to the bovine strain of the tubercle bacilli, and 2 c.c. injected into the pectoral subcutaneous tissue of a chicken, whose greatest susceptibility is to the avian strain of the tubercle bacilli. The latter two animals remained perfectly normal, the rabbit being autopsied at the end of six weeks and did not show any lesions at all. The chicken remained perfectly well and was not posted. The guinea pig died within five weeks after injection, and showed a caseous inguinal lymph gland, which had ulcerated externally through the skin. Smears from this gland showed tubercle bacilli. Lesions of a tuberculous nature were also demonstrable in the lung and the spleen. Smears were not made from the latter areas. Sections taken from the glands, spleen and the lungs showed tubercles typical in all respects.

A portion of the left cervical gland (see Fig. 7) was treated in the same manner as the above. Two c.c. were injected into the inguinal region of a guinea pig. At the end of six weeks the pig was killed and upon autopsy showed no lesions.

Six months after glands of Figure 7 were removed, another left cervical gland was removed. Grossly and microscopically it was similar to that of Figure 7.

Sterile glandular material of the second group of the left side was treated as above with the same negative results.

HEMATOLOGIC STUDIES.

	Apr. 15, 1929	Apr. 17, 1929	Nov. 30, 1929	Nov. 30, 1930
R. B. C.....	4,320,000			3,820,000
W. B. C.....	12,600	6,700	7,200	9,650
Polys.....	68	57	65	62
L. L.....	5			
S. L.....	21			
Eosin	1	3		
Baso	1		1	
Lymph.....		39	32	35
L. M.		1		

The anemia is of the secondary chlorotic type, and may become severe or pernicious. The leucocytes are usually under 10,000; often there is leukopenia, occasionally leucocytosis of 30,000 or more. Relative or absolute lymphocytosis usually prevails, but the neutrophile cells are increased during the exacerba-

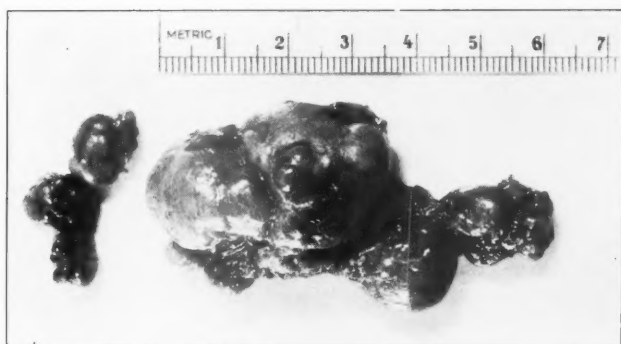


Fig. 5. Photograph of enlarged right cervical lymph glands, each apparently preserving its individuality, but held together by fibrous tissue, which were removed 29 days after the gland from the right posterior pharyngeal wall. Through the delicate intact capsule could be seen a grayish pink, translucent soft parenchyma.

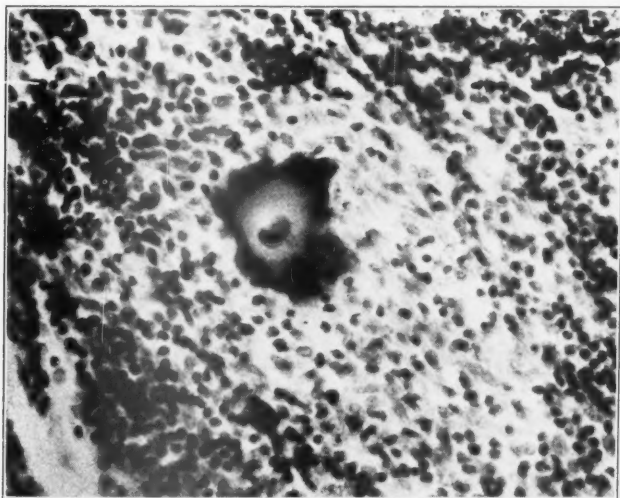


Fig. 6. Section of right cervical gland. Presented a tuberculous giant cell surrounded by epithelioid and lymphoid cells forming an early tubercle. Diagnosis: Hyperplastic tuberculosis.

tions. Eosinophilia is so frequent and marked as to form an important diagnostic sign. Mast cells may be increased.

CLINICAL COURSE.

The clinical course of Hodgkin's disease varies from an acute variety which ends fatally, in from four to six weeks, to the usual chronic type, which, starting as a benign enlargement of the cervical nodes, extends eventually to all the lymphoid tissues of the bodies. Hodgkin's disease occurs often in childhood, males being more frequently affected than females. When occurring in children, throat infections are common and the initial pathology being presented in the cervical nodes. At first the nodes are limited to one side, but later the opposite chain of cervical nodes is affected. By progressive extension to the neighboring glands, the disease spreads to all the lymphoid tissues. The enlarged nodes remain discrete, though their combined bulk may present a large tumor mass. The skin over the glands itches, but seldom becomes involved unless a secondary infection of the glands occurs. The tonsils are nodular, and in rare instances there occurs ulcerations of the larynx, with enlargement of the adjacent glands. Eventually the entire lymphatic system is involved and the end comes with a cachexia and secondary anemia.

Mikulicz's disease is a specialized type, in which the pathology begins in the salivary or lacrimal glands. The subsequent course remains the same.

Lesions of the mucous membrane of the mouth and tongue may be the earliest visible manifestation of Hodgkin's disease. Histologically these lesions will show infiltration of the subepithelial layer of the membrane with endothelial and epithelioid cells, eosinophile and histiocytes.

Sternberg contended "that Hodgkin's disease was an atypical type of tuberculosis, because in a series of fifteen cases pseudo-leukemia he was able to demonstrate tuberculous lesions in one or more organs in eight of the patients." Since his article, pathologists have argued the pro and con of this dispute with the accepted idea today being that Hodgkin's disease is not of tuberculous origin

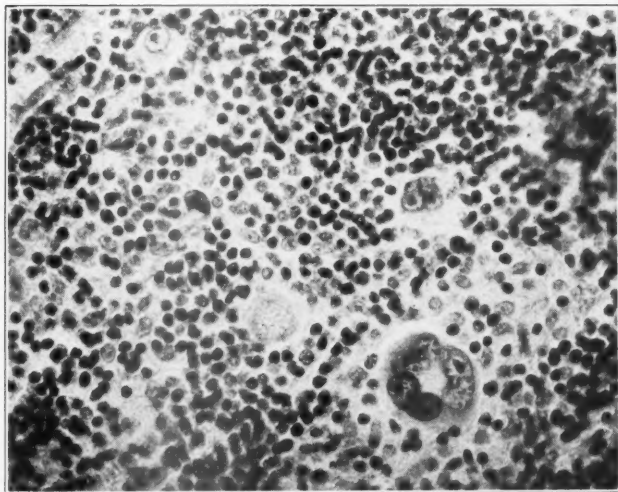
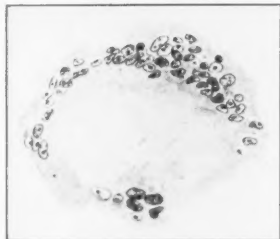
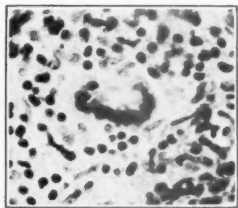


Fig. 7. Section of left cervical gland removed 11 months after right cervical glands. Showing endothelial cells, lymphocytes, eosinophiles, mitoses, and a number of Dorothy Reed giant cells. Diagnosis: Hodgkin's disease.

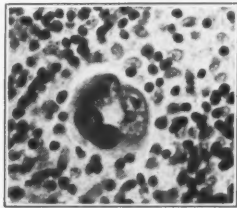
GIANT CELLS



Foreign Body



Langhans



Reed

Fig. 8. Showing a typical Dorothy Reed and a Langhans' giant cell selected from the biopsies and contrasted with a foreign body giant cell.



Fig. 9.

Taken May 3, 1932, showing enlargements apparently under control.

Our young girl had glands showing each variety in close proximity. Ewing reports a case of phthisis in which three small adjoining bronchial nodes showed, miliary tubercles in one, a diffuse lymphocytosis in the second and typical Hodgkin's granuloma in the third. ("Neo-Plastic Disease," James Ewing, published by W. B. Saunders & Company, page 407.)

LYMPHOSARCOMA.

Levin contends "through years of parallel study of lymphoma malignum and lymphosarcoma on the one hand and of all clinical types of carcinoma and sarcoma on the other, I have gained the conviction that lymphoma malignum (Hodgkin's disease) and lymphosarcoma are nearly identical in their clinical manifesta-

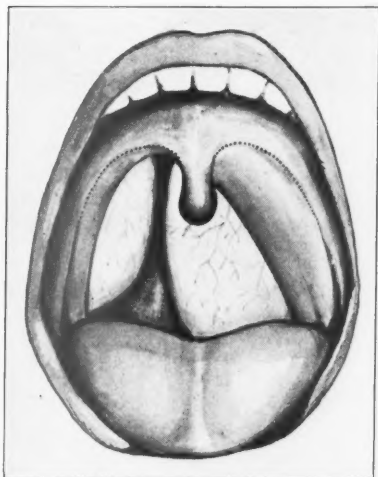


Fig. 10. Illustration of the enlarged pharyngeal glands.

tions, that they both represent a special type of malignancy and that the peculiarities in the structure and clinical manifestations of lymphoma malignum (Hodgkin's disease), as compared with other types of malignancy, depend not so much on the difference in the causation but on the special character of the structure and function of lymphoid tissue.

Malignant lymphoma (Hodgkin's disease) also begins as a swelling of one group of lymph nodes. Microscopically, there is noted a hyperplasia of all types of cells of a normal lymph node—the large and small lymphoid cells, the reticulum cells, as well as frequently the granulated leucocytes, mainly eosinophils. There is also present a peculiar type of a giant cell (Dorothy Reed cell). Thus, morphologically, the condition resembles rather an inflammatory granuloma than a malignant tumor.

Microscopically, the main feature of malignant lymphoma is not a degenerative process as in syphilis or tuberculosis, but an active proliferation of all types of cells of the lymph nodes.

Lymphosarcoma differs microscopically from malignant lymphoma, as it usually shows the proliferation of only one type of

the cells of the lymph node—either the reticulum cells or the lymphocytes. Furthermore, in lymphosarcoma the cells break through the capsule of the lymph node and invade the adjoining tissue. This, however, may also take place in lymphoma malignum."

GIANT CELLS.

Histologically, the enlarged nodes of Hodgkin's disease present endothelial and epithelioid cells, small and large lymphocytes, fibroblasts, Dorothy Reed giant cells, eosinophiles and histiocytes. These giant cells have one or more nuclei grouped in the center of the cell with a moderate amount of cytoplasm.

Foreign body giant cells contain cholesterol crystals and a large mass of cytoplasm with many small nuclei.

TREATMENT.

The value of roentgen therapy is well illustrated in this case, as our little lassie has grown and maintained a good state of general health.

CONCLUSION.

1. Hodgkin's disease and tuberculosis of the lymph glands are separate pathologic entities.

2. The enlarged nodes of Hodgkin's disease are radio-sensitive.

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Pathological and bacteriological studies were made by Harry C. Schmeisser, M. D., Director of the Department of Pathology, University of Tennessee, College of Medicine.

Illustrations were made by the Pathological Institute, University of Tennessee.

LXIV.

VIDIAN NEURALGIA.*

HARRIS H. VAIL, M. D.,

CINCINNATI.

Vidian neuralgia is a pain of neuralgic character in the nose, face, eye, ear, head, neck and shoulder, occurring in severe attacks. These cannot be caused by any external stimulation, are not relieved by opiates and are not associated with any subjective loss of sensation. The attacks are most typically unilateral, are often nocturnal and may or may not be associated with the subjective symptoms of a nasal sinusitis.

Greenfield Sluder¹ described as sphenopalatine ganglion neuralgia, pain in the root of the nose and in and about the eye, in the upper jaw and teeth (sometimes lower jaw and teeth), extending backward under the zygoma to the ear, frequently making earache and pain in the mastoid, but severest often at a point 5 cm. back of the mastoid; extending thence to the occiput, neck, shoulder blade, shoulder, breast, and when severe, to the arm, forearm, hand and fingers; with sometimes a sense of sore throat on that side. Rarer additions to this picture are itching of the skin of the upper extremity, taste disturbances, a sense of stiffness and muscle weakness in the upper extremity and fortification scotomata. It may appear as a constant pain with exacerbations, or it may stop and reappear as a migraine, or it may stop and reappear with the stabbing sharpness of a tic. Motor phenomena are changes in the appearance of the soft palate—i. e., the arch is higher on the affected side. The uvula is inclined obliquely to the well side. There may be a slight blunting of the tactile sensation of the soft palate, pharynx and tonsils with a like condition of the membrane of the nose of the affected side. Sluder stated he had never seen all the above manifestations in any one case. He stated that he was able to produce, by electrical stimulation

*Presented in the Instruction Course at the thirty-sixth annual meeting of the American Academy of Ophthalmology and Otolaryngology at French Lick, Ind., September 13-19, 1931.

of the vidian nerve in the sphenomaxillary fossa, pain in the ear, mastoid, neck, shoulder blade, arm and forearm. He felt that the syndrome described by him as sphenopalatine ganglion neuralgia might be produced by lesions of the nerve trunks which supply the ganglion and offered a method of differential diagnosis by means of cocaineizing the sphenopalatine ganglion which should stop the pain if the lesion were in the ganglion but would have no effect upon the pain if it were a case of vidian neuralgia.

He reported several cases which he considered vidian neuralgia, and described tic of the vidian nerve as sharp, recurring, stabbing attacks of pain in the lower part of the head at intervals of not more than a few hours apart.

The literature on Meckel's ganglion neuralgia is a very extensive one, but apparently that of vidian neuralgia, with the exception of the work of Sluder and Bliss,³ had been absent from the literature until 1929, when I⁴ reported a case of sphenoiditis which showed not only the posterior symptoms hitherto assigned to irritation of the vidian nerve, but also the anterior symptoms which Sluder felt were due to irritation of Meckel's ganglion. Since that time I have felt that Sluder was in error when he attempted to differentiate between sphenopalatine ganglion neuralgia and vidian neuralgia, and I shall hope to convince you that the painful symptoms in the condition under discussion are due to an irritation or inflammation of the vidian nerve and not to any conditions affecting directly the sphenopalatine ganglion.

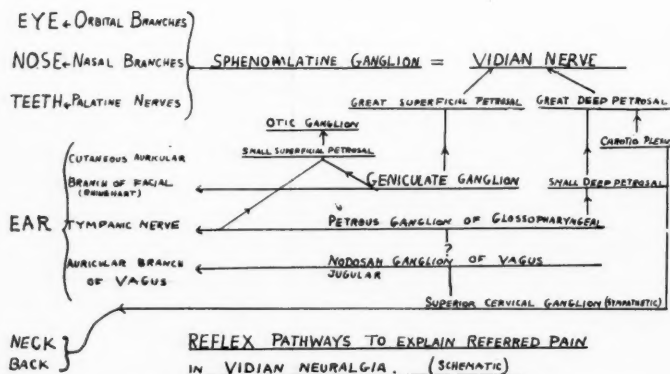
Sluder,⁵ under the heading of his first ganglion case, recites the history of a man, 45 years of age, who was first seen in 1903. For ten years he had suffered headaches which incapacitated him for business two or three days of almost each week. The pain began at the root of the nose on the right side, taking in the upper jaw and extending backwards to become emphasized at the tip of the mastoid for a distance of 5 cm. posterior to it. The attacks were not always of equal severity. In severe attacks the pain extended to the neck, shoulder and shoulder blade of the same side and lasted from two to twenty-four hours. Another case was that of a woman, 27 years of age, who had suffered for many years, off and on, paroxysmal attacks of pain beginning at the root of the nose, involving the upper jaw and teeth, ex-

tending backward to the tip of the mastoid; with each attack of suppurative posterior sinusitis the pain recurred and spread into the neck, shoulder, shoulder blade and arm in great severity. Neither of these cases were classed by Sluder as vidian neuralgia. Comparing the symptoms of the cases which I report here with the cases reported by Sluder, it will be seen that they are identical. Even if we disregard for the moment the question as to whether the condition should be called vidian neuralgia or sphenopalatine neuralgia, there is this definite syndrome shown by all the cases, which I shall describe again as being stabbing, severe paroxysmal attacks of pain that start on one side of the nose, radiate into the eyeball, forehead, upper jaw, ear, neck, back of the head, and shoulder on the same side.

Now I shall attempt to show that this condition is due to an irritation or inflammation of the vidian nerve in the vidian canal, and is always secondary to a latent or frank infection in the sphenoid sinus. Sluder⁴ states that the clinical fact remains that certain sphenoidal inflammatory cases simulate completely the typical neuralgic and sympathetic phenomena arising in the nasal ganglion.

It is quite obvious, of course, that the only rational treatment for this condition depends upon the exact etiology, and I shall try to convince you that treatment directed towards the sphenopalatine ganglion is not the proper kind. To make my point clear, we must consider in detail the anatomic relations in the region under study.

First of All the Vidian Nerve.—This nerve is formed by the junction of the great superficial petrosal nerve and the great deep petrosal nerve at the beginning of the cranial opening of the vidian or pterygoid canal of the sphenoid bone. The vidian nerve passes through the vidian canal to end in the sphenopalatine ganglion. The vidian nerve does not enter into the sphenomaxillary fossa, as the sphenopalatine ganglion sends a prolongation a short distance into the vidian canal. This fact is of prime importance, as it would be impossible by an electrical stimulation or alcohol or novocain injection in the sphenomaxillary fossa to act upon the vidian nerve without acting upon a portion of the sphenopalatine ganglion. For this reason, Sluder's experi-



This chart shows the pathway of reflex pain from stimulation of the vidian nerve. Impulses to the front of the head pass from the vidian nerve to the sphenopalatine ganglion and from there to the eye, nose and teeth. Impulses pass backwards through the great superficial petrosal nerve and the great deep petrosal eventually to reach the ear, back of the head, neck, back and shoulder.

ments made, as he said, by electrical stimulation of the vidian nerve in the sphenomaxillary fossa of a living patient must be disregarded.

The recent studies of the sphenopalatine ganglion by Dr. Klaus Vogel of Berlin⁷ show that the vidian nerve is completely interrupted in the sphenopalatine ganglion.

Thus, the ganglion is the ending of the vidian nerve. Many anatomic specimens show a very close relation between the vidian nerve in its canal and the mucosa of the floor of the sphenoid sinus—in fact, the relation between the nerve and the sinus is usually much more intimate than that between the sphenopalatine ganglion and any of the sinuses about it. About the sphenopalatine ganglion there is considerable areolar fatty tissue and the ganglion is held loosely in a comparatively large space. After comparing these facts with the known relations of the vidian nerve to the sphenoid sinus it is logical to feel that an inflammation in the sphenoid sinus would be much more apt to cause inflammatory reaction in the vidian nerve, where it is held in a bony canal, than it would the sphenomaxillary fossa, thus indirectly acting upon the ganglion.

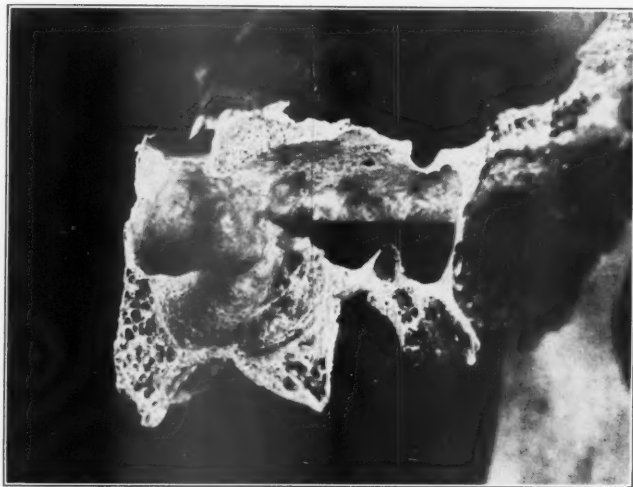


Fig. 1. This is a photograph of an anatomical specimen to show the picture obtained by filling of the sphenoid sinus with radio opaque material and an X-ray made with the head in a vertical position, the beam of the X-ray being passed anterior posteriorly. The vidian canal projects upwards into the lumen of the sphenoid sinus. Quite similar pictures were obtained on actual patients.

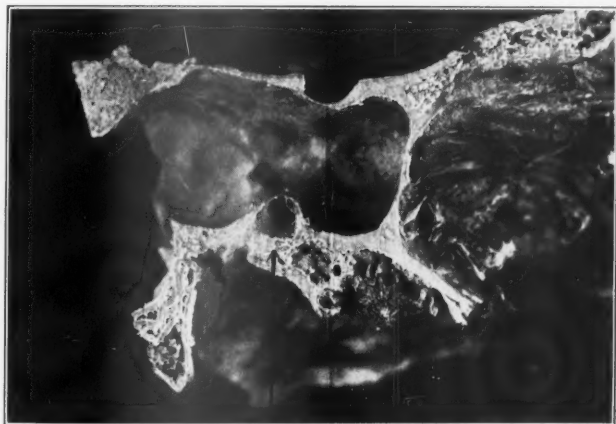


Fig. 2. A photograph of an anatomical specimen showing the vidian canal projecting upwards into the cavity of the sphenoid sinus with slight pneumatization of the pterygoid plate.

Vogel⁸ has shown there exists in atrophic rhinitis a widespread atrophy of the sphenopalatine ganglion with marked connective tissue formation about the ganglion cells and a great disappearance of the latter. This is one condition where inflammation of the sphenopalatine ganglion has been proven, and yet it is exceedingly rare to find any symptoms of sphenopalatine or vidian neuralgia in cases of atrophic rhinitis.

Vogel⁹ and Schatz¹⁰ have reported cases which showed no secretory and only transient sensory disturbances in the nose after the removal of the sphenopalatine ganglion.

Moulouguet and Collin¹¹ removed in dogs the sphenopalatine ganglia without any sensory or trophic disturbances. They quoted the work of Jung, Tagand and Chavanne,¹² who stimulated the vidian nerve in dogs and obtained a copious nasal secretion. After section of the vidian nerve there was diminished nasal secretion.

Infection and tumors in the sphenomaxillary fossa can produce painful neuralgia symptoms. The same symptoms sometimes follow blocking of the sphenopalatine ganglion for local anesthesia in the radical antrum operation. These conditions are different from vidian neuralgia.

Ullman¹³ reported a case history of a patient who, eight days after a radical antrum operation under local anesthesia where the technic of novocain injection was that of Slobodnik, showed massive swelling of the cheeks with severe neuralgia pains in the teeth, region of the ear and temple. All the symptoms increasing, a wide incision for phlegmon of the neck was done, with ligation and resection of the internal jugular and facial veins. Eventually recovery ensued. He reported three similar cases which were fatal, two of which were injected after the method of Slobodnik.

To explain the distribution of the painful symptoms in vidian neuralgia, we find that the great superficial petrosal nerve comes from the geniculate ganglion of the facial nerve; the great deep petrosal is formed by the branch from the carotid plexus and the small deep petrosal from the petrous ganglion of the glossopharyngeal. Thus through the nerves uniting to form the vidian nerve there is a close tie-up between the sphenopalatine ganglion, the geniculate ganglion of the facial nerve, the superior cervical

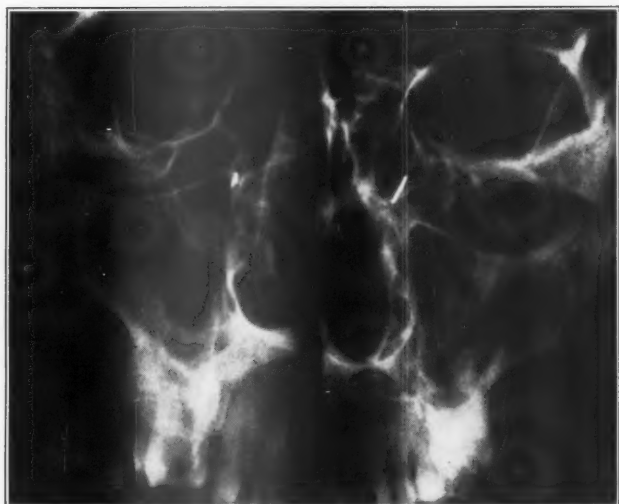


Fig. 3. A half of two cadaver's skull with a copper wire inserted in the vidian canal. On the left side the X-ray beam strikes the copper wire more nearly end on than on the right side. This localizes the vidian canal with respect to the lateral wall of the nose. Anteroposterior view.

sympathetic ganglion, the glossopharyngeal nerve and the vagus nerve. From the sphenopalatine ganglion pass orbital, nasal and palatine branches, so that it is very easy to explain the referred pain in the eye, nose and teeth. The explanation of the pain in the ear, a very typical and characteristic part of the vidian neuralgia syndrome, is a little more complicated. Fenton and Larzell¹⁴ have made an intensive study of the sphenopalatine ganglion and its connections, and feel that the pathway of reflex otalgia from involvements of the sphenopalatine regions is through the great superficial petrosal nerve to the geniculate ganglion and out through the ramus cutaneous of the facial (nerve of Rhinehart) to the auricular and mastoid region.

Apparently the existence of the cutaneous branch of the facial nerve is admitted by many anatomists, though not definitely found in man. In herpes oticus there are strong clinical evidences of its existence.



Fig. 4. A half of a cadaver's skull with a copper wire in the vidian canal, showing its relations to the sphenoid and the lateral wall of the nose.

One of the two other sensory nerves to the ear, namely, the tympanic nerve, arises from the petrous ganglion of the glossopharyngeal and runs through the middle ear cavity to supply the mucosa of the mastoid and eustachian tube. It is joined by a branch from the geniculate ganglion to form the small superficial petrosal nerve which ends in the otic ganglion. In this manner the geniculate ganglion not only can influence the cutaneous branch of the facial nerve, but also the tympanic nerve from the glossopharyngeal. Besides the tympanic nerve there is the auricular branch of the vagus from the nodose ganglion, which is united with the superior superficial cervical ganglion, and the petrous ganglion of the glossopharyngeal.

The pain in the ear described by sufferers with vidian neuralgia is due to impulses passing backward through the great super-

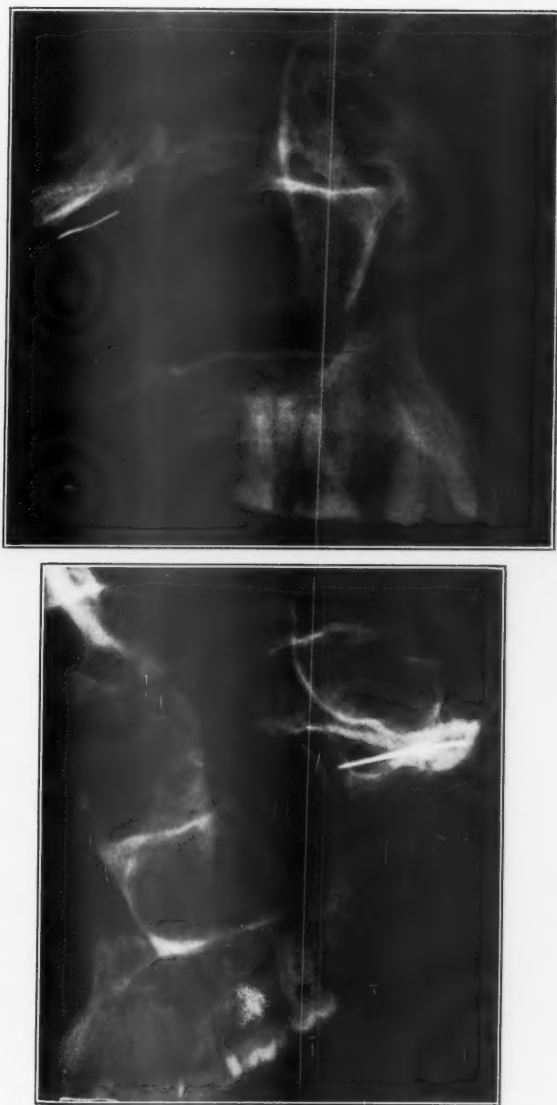


Fig. 5. Lateral X-ray view of the half of two cadaver's skulls with copper wire inserted in the vidian canal. In the lower picture the wire is passed into the sphenomaxillary fossa. This will show the angle of which the vidian canal passes.

facial petrosal nerve to the geniculate ganglion, and from there by either the cutaneous branch of the facial or by the tympanic nerve, while a more roundabout pathway is through the auricular branch of the vagus.

The pain in the neck, back and arm can be explained by impulses passing backwards through the great deep petrosal nerve to the superior cervical sympathetic ganglion and from there to the cervical nerves.

With such a close relation between the vidian nerve and the sphenoid sinus as can be seen in many specimens, and by the fact that a sphenoiditis can be present in a great many cases without giving obvious signs of its existence, and by knowing that relief of the painful symptoms has been obtained by treatment of the sphenoiditis, it would seem fair to feel that the syndrome described by Sluder under the heading of sphenopalatine ganglion neuralgia and described by me under the heading of vidian neuralgia is really the same syndrome and that its etiology should be looked for in the vidian nerve

As for diagnosis and treatment, we know that a sphenoid infection can be latent and will not show evidence of its existence unless a very careful search is made. A routine examination is not sufficient in these cases. One must shrink up the posterior part of the nose, look carefully for mucoid secretion, for the presence of atrophy of the membranes in the back part of the nose, for obstruction from a deviated septum and enlarged middle turbinates. In all cases it is advisable to either probe the sphenoid sinus or, better still, to fill it with radiopaque oil for roentgen ray study. Sometimes filling defects in the sphenoid sinus will be noted. In all cases the size of the sinus and its relation to the vidian canal can be shown. Careful histories are essential.

A differential diagnosis should include trifacial neuralgia, otalgia dentalis, tumors of the nasopharynx, disease of the apex of the petrous bone and neuralgia directly due to acute or chronic anterior sinusitis.

Once a diagnosis has been made, if the case is a mild one or of fairly recent duration, an injection of the sphenoid sinus with



Fig. 6. X-ray of the half of a cadaver's skull with wire in the vidian canal passing into the sphenomaxillary fossa and a longer probe inserted through the sphenopalatine foramen in the technique used for alcohol injections of Meckel's ganglion. This shows that the axis of the sphenopalatine foramen and the vidian canal is on the same plane.

lipiodol following the use of ephedrin, nasal douching and colloidal silver preparations often will obtain relief. Many acute attacks can be controlled by inserting a pledget of 10 per cent cocain without adrenalin in the sphenoid recess against the front wall of the sphenoid sinus and allowing the patient to recline with the pledget in the nose for fifteen or twenty minutes.

Severe cases require a surgical procedure. This should consist of a submucous resection of the septum, which is carried back to the sphenoid, the necessary trimming of the middle turbinate and the fullest exposure of the sphenoid with an opening in the frontal wall of the latter, so that its cavity may be inspected and any pathology there dealt with. This is to be followed by post-

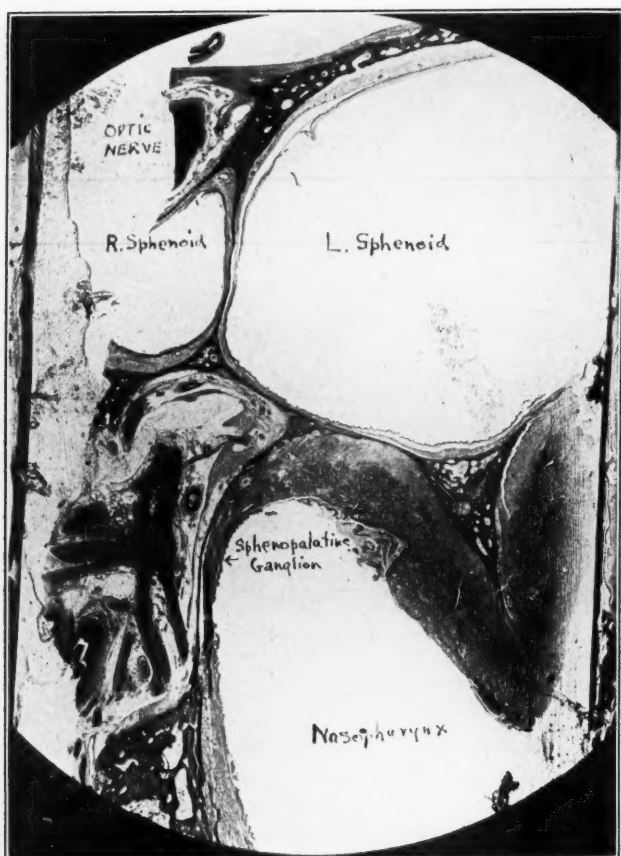


Fig. 7. A photomicrograph of a slide obtained through the kindness of Dr. Klaus Vogel of Berlin. (Magnification 8 to 10 times.)

This shows the sphenomaxillary fossa with the sphenopalatine ganglion and the sphenoid sinus. One sphenoid is very large and projects beyond the midline.

From a study of this section it is rather difficult to see how a sphenoid sinus suppuration could affect directly the sphenopalatine ganglion. There is considerable tissue between the ganglion and the surrounding bone and the ganglion is held loosely in the supporting tissue. In addition the ganglion has a definite capsule which is not connected in any way with the submucosa of the sinuses.

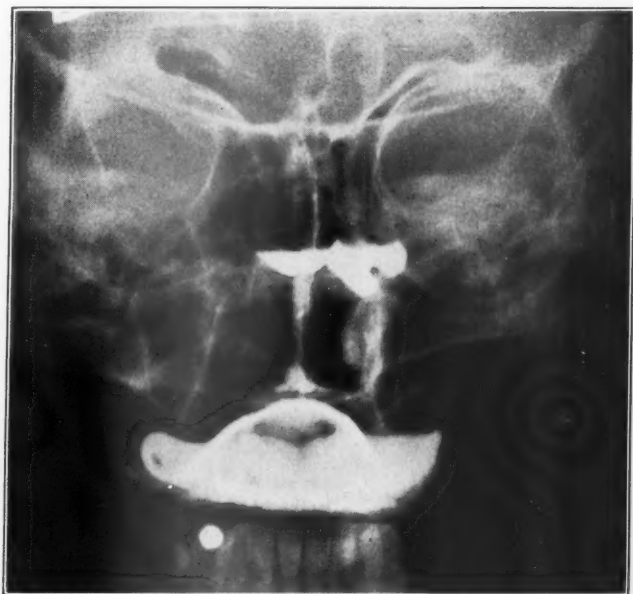


Fig. 8 (Case Report). Mrs. W. E. S., age 54, for four years with each cold has severe pain starting at the root of the nose on the right side, passing in and over the right eye into the temple and back of neck. Pain never in ear. Attacks come about every two weeks, lasting one to two days, often awakening her from 2 to 3 o'clock A. M. The right side of nose stopped up.

Lipiodol injected into the right sphenoid, anterior-posterior view with head in the vertical position shows the vidian canal projecting like a peninsula into the sinus. Attacks very much relieved by local treatment to the nose.

operative treatment for a period of weeks directed to subdue the inflammation in the sphenoid sinus. At the time the sphenoid is opened the posterior ethmoid cells should be also opened.

The prognosis which is, as a rule good, depends upon the duration and severity of the symptoms, the degree in which the sphenoiditis can be eliminated and the proximity of the vidian nerve to the sphenoid sinus. When the vidian nerve is lying in the floor of the sinus with a very thin covering of bone, it will be affected more severely by a milder infection than a better protected nerve. Recurrences of the pain are to be expected; they come whenever there is an infection in the sinus and last as long



Fig. 9 (Case Report). Mrs. E. B., age 45, for the past five years has had severe attacks of pain starting below the right eye along side of the bridge of the nose, passing over the eye, in the back of the head, into the ear, down the neck, back and shoulder—worse with colds.

Examination of the nose showed a very badly deflected septum. Impossible to probe the right sphenoid. Sphenoid operation done followed by great relief and no pain until over a year later when there was a return of the pain; promptly relieved by local treatment to the sphenoid.

Lipiodol was injected into the sphenoid sinus after operation and to hold it in place a pledget of cotton was inserted. This can be differentiated from the lipiodol in the sphenoid.

as the infection continues. A satisfactory sphenoid operation makes it much easier to control subsequent infections and to relieve the severe attacks by injecting a few drops of cocain solution into the sphenoid sinus.

The incidence of vidian neuralgia is greater than one would suspect. In my private practice I have seen 31 cases diagnosed as such, of which 28 were females. The youngest patient was 24 years old, the oldest was 59 years.

A tabulation of the cases shows the following:

CASES OF VIDIAN NEURALGIA—TOTAL, 31.

Males.....							3
Females.....							28
Ages.....	10-19	20-39	30-39	40-49	50-59	60-70	
	0	5	6	13	7		



Fig. 10 (Case Report). Miss D. W., 28 years of age, for eight years has had attacks of pain starting on the right side of the nose, passing into the right eye and top of the eye, into the temple, zygoma, ear, down the back of neck and head. There are periods of freedom for several months. When the pain returns it may last constantly for two weeks. Always starts with a cold. She notices dripping in the nose, throat gets thick and then the pain starts. Relief of severe attacks was always obtained by cocainizing the front wall of the sphenoid sinus.

Lipiodol injected into the sphenoid shows a slight pneumatization of the pterygoid plate.

Of this series, nine patients were operated upon with relief in eight—one operated case showed considerable improvement for the few weeks she was under my care—later reports were that she was as bad as ever. The remaining cases were sufficiently relieved by local treatment so that they were satisfied.

Stewart and Lambert¹⁵ reported a series of 79 cases of neuralgia under the diagnosis of lower half headache, migraine, sphenopalatine neuralgia, sphenopalatine neurosis or syndrome tic douloureux, sympathetic pain, maxillary and vidian neuralgia. Of the 79 cases, 59 were females, of which 9 were between the ages of 20 and 29—19 between the ages of 30 and 39, and 11 between the ages of 40 and 49. Their treatment consisted of cocainization

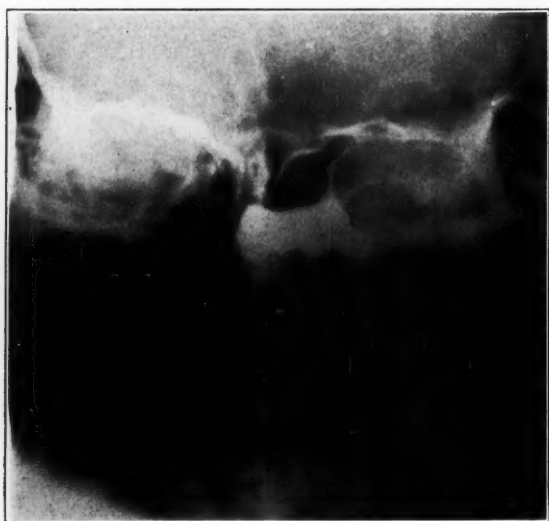


Fig. 11 (Case Report). Miss M. C., age 45, for several years has had attacks of severe pains starting in the right side of the nose, passing into the eye, cheek, ear, back of neck, down the neck, into the shoulder and arm. Attacks last an hour or so, come night and day at no special time, though worse in the afternoons and evenings. She has had considerable pansinusitis with a double radical antrum, ethmoid and left external frontal sinus operation.

Lipiodol injected into the right sphenoid shows a large sinus with extreme pneumatization of the pterygoid plate. The vidian canal can not be seen.

Operation upon the right sphenoid—nasopharyngoscope passed into the sinus showed the ridge of the vidian nerve. It was sharp and permanent with a deep recess on either side. No differentiation in color. Marked improvement followed operation but recurrences of pain with attacks of sinusitis. The pain is always relieved very quickly by treatment to the sinusitis.

with relief in 43 cases, alcohol injection with complete relief in 15 cases, incomplete in 6 cases and no relief in 9 cases. They felt that there existed a neuralgic type of pain of widespread distribution about the head and neck which differed from true *tic douloureux* and that its cause was probably due to nasal infection but that its underlying neurologic nature was obscure.

I can only speculate why so many more females should be affected by vidian neuralgia than males. A check-up of the histories of my patients showed that some had had the neuralgia for



Fig. 12 (Case Report). Mrs. G. P., age 46, for three years has had neuralgia pain on the right side, starting over the right eye, passing into the cheekbone, ear, roof of mouth, back of head and down the neck. Attacks have increased in severity so that for the past month they come every two or three hours, lasting fifteen or twenty minutes. Has not been subject to colds but has had postnasal discharge and nasal obstruction. A year ago had a submucous resection of the septum. X-ray of the sinuses were reported clear.

Lipiodol injected into the right sphenoid and antrum with X-rays showed no filling defects. On the above X-ray film the arrows indicate the vidian canal.

Attacks of pain were relieved by injection of cocaine into the sphenoid sinus. At the sphenoid operation there was polypoid thickening of the floor of the sphenoid. Following operation pains occurred at four to six hour intervals for several days, improving gradually to one attack in twelve hours. By two weeks after operation the pain had left the top of the head, ear and eye, and was much milder throughout the head. Later the pains returned but further details of the case are not known as patient did not return.

a relatively short time, while others had had it for years. In no case was it present under twenty years of age. This fact is consistent with our knowledge as to when the sphenoid sinus develops to its fullest extent. That vidian neuralgia is not a disease of the menopause is also obvious. However, the greatest number of my patients were at this stage, and as a result their symptoms had been laid by their local or family physicians to it—that this was not so was manifested by the relief obtained from local or operative treatment to the sphenoid sinus.

Another observation made from a study of the case histories was the frequency of attacks at 2 to 3 a. m. Just why this should be I cannot say. Ordinarily, nocturnal attacks of neuralgia pain should make one think of a luetic infection, yet in none of my patients was there any lues, clinically or serologically.

SUMMARY.

1. It would seem that there is a definite clinical syndrome which has been described as sphenopalatine ganglion neuralgia and vidian neuralgia, and which would seem from anatomic and clinical studies to be due to an irritation or inflammation of the vidian nerve. For this reason it seems proper that the term vidian neuralgia should be applied to this syndrome rather than the term sphenopalatine ganglion neuralgia (Meckel's ganglion). The irritation in the nerve can pass either to Meckel's ganglion, from there on out to the front of the face, or it can pass backwards to the geniculate ganglion and from there to the back part of the head and ear.
2. Anatomic facts are quoted to refute the experiments of Sluder and to show that it is impossible to stimulate the vidian nerve in the sphenomaxillary fossa without involving the sphenopalatine ganglion.
3. The condition is one of adult life and is most frequently found in females.
4. Brief case reports with roentgen ray films are presented.
5. The treatment should be directed towards the disease in the sphenoid sinus.



Fig. 13 (Case Report). Sister E., age 51, for ten years has had sinus trouble. Bilateral antrum, ethmoid and sphenoid operations had been performed. In the past three years has had attacks of pain starting above and in the right eye and in the back of the head on the left side. The right sided attacks start along side of the bridge of the nose, get back of the eye, into the eyeball, and make her feel that the eye is being pushed out. The pains never get back as far as the ear. Pain on the left side starts in back of the head, travels to the back of the ear, above and into the ear, through the temple to end in the forehead. Never has the pains on both sides at the same time. Attacks come with colds.

Examination of the nose showed adhesions closing up the right sphenoid sinus. After these were relieved irrigation of the sphenoid evacuated pus.

Lipiodol injected into both sphenoid sinuses but did not stay in the right sphenoid. The oval shadow apart from the filling of the floor is a pledget of cotton packed against the operative opening of the sinus.

At a bilateral sphenoid operation, pus and polypi were found in the right sphenoid, none in the left. Upon removing a polyp in the outer angle of the right sphenoid patient complained of a severe sharp pain in the right eye, typical of her attacks. This was followed by complete relief of this pain and it never has recurred during the two years that the patient has been under observation. She has had some attacks of pain on the left side and this can be produced at any time by probing the floor of the left sphenoid. With recurrences of symptoms the left sphenoid has always been inflamed. This is one of the few bilateral cases.

24 EAST EIGHTH ST.

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LXV.

EXPERIMENTAL EDEMA OF THE LARYNX PRODUCED BY PARAPHENYLENEDIAMINE.

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ST. LOUIS.

An adequate explanation for the sudden onset of edema of the upper respiratory tract, such as angioneurotic edema of the larynx, has never been offered. However, it has been suggested that this phenomenon may be related to allergy and is probably due to increased permeability of the blood vessels.¹

While searching for experimental methods that might throw more light upon the problem of the formation of edema of the upper respiratory tract, a chemical compound, paraphenylenediamine, was found worthy of further investigation. This substance first attracted considerable attention when used as a fur and hair dye, for in susceptible individuals it produced an irritation of the skin, dermatitis, swelling of the eyelids and face, and sometimes brought on attacks of asthma.

Dubois and Vignon² were the first to point out that its administration to dogs causes edema of the head and neck, and sometimes exophthalmos. Other animals, such as the rabbit and cat, were similarly affected. The effect of paraphenylenediamine on rabbits and cats has been described in detail by Gibbs³ and by Tainter and Hanzlik.⁴ The effect in rabbits is very striking. Oral or subcutaneous administration in doses of .1 to .2 grams per kilo causes edema of the face and sometimes exophthalmos. The tongue becomes markedly edematous and protrudes from the mouth. The buccal cavity likewise becomes edematous to such an extent that the jaws cannot be closed. The glottis and vocal cords become acutely edematous, and the vocal cords come in apposition. Death results from asphyxia unless a tracheotomy is performed, which prolongs the

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life of the animal for several hours. All of these changes appear about an hour and fifteen minutes after the administration of the drug.

Gibbs found that the edema appears in other parts of the body, but the vessels of the head and neck seem to be the most affected by the drug. Upon incising the edematous areas some serum escapes, but the tissues do not collapse, and the wound gapes. The effect is somewhat similar to making an incision in a nasal polyp. The mechanism of the reaction is apparently due to an increased permeability of the blood vessels of the head and neck, thus permitting fibrin, one of the least diffusible of the blood proteins, to pass through the vessel walls. Gibbs has shown that this permeability change is independent of the central nervous system, since it occurs in decerebrate animals. Electrical stimulation of the superior cervical ganglion retarded, but did not prevent the formation of edema on the corresponding side. This delayed response probably was brought about by a decreased concentration of the drug. This was confirmed by Tainter and Hanzlik, who also demonstrated that vasodilators, such as nitrites, accelerated the effect, and vasoconstrictors, such as adrenalin, retarded the formation of the edema. Tying off the carotids and thus blocking off the access of the drug to the head and neck region, prevented the edema from forming.

No marked effect on the blood pressure was noted. A slight increase in concentration of the blood was found, probably due to loss of fluid. An interesting finding was the marked increase of fibrin content of the plasma. In normal animals Gibbs found the clotting time to be from 34 to 49 seconds, but after one and one-quarter hours had elapsed after administration, the clotting time was too rapid to be determined.

Another interesting finding of Gibbs was the production of edema in the corresponding head and neck areas similar to paraphenylenediamine, by perfusing the ascending aorta with Ringer's solution. No kidney changes were found, while oxidative processes and acidosis seemed to play no part. Paraphenylenediamine is readily diffusible, and was found in equal concentration in the tissues and in the blood. No direct effect, such as colloidal inhibition or swelling, was detected. Certain dyes, such as Congo red,

which do not pass through normal blood vessels, escaped into the surrounding tissue spaces of rabbits after receiving the drug. A small percentage of rabbits do not react in the typical manner, and this is explained by the lack of sufficient blood pressure. Localized edema seems to be dependent on such features as relative vascularity, permeability of capillaries and capillary pressure and the amount of mechanical support. Gibbs demonstrated that the action of paraphenylenediamine on the blood vessels of the head and neck is quantitative rather than qualitative. He demonstrated that if the blood vessels of a limb were kept in tone, the injection of the drug into the nutrient artery caused edema to form. He points out that the effects of paraphenylenediamine and histamine are similar, but that the former is weaker. Both substances caused increased capillary permeability and constriction of the arterioles. Unless histamine is given in minute doses the effects on the intestine mask the other vascular reactions.

In this connection it is of interest to mention the reports of Lewis,⁵ who found that in the human the blood vessels of the head and neck are susceptible to histamine more than elsewhere. Paraphenylenediamine is much less active than histamine, and when given in moderate doses acts like very small doses of histamine. Why some capillaries are more susceptible to this action than others cannot be explained. Gibbs made interesting observations on the capillaries of the lung, which seem highly resistant to changes in permeability when the oxygen supply is adequate.

Five rabbits, under sodium amytol anesthesia, were given subcutaneously 0.2 gms. per kilo of paraphenylenediamine base (Eastman Kodak Co.) dissolved in about 20 cc. of distilled water. One rabbit died without typical edema formation, while the remaining four, after an interval of one hour and a half, reacted as described above. The edema was confined to the head and neck area. Tracheotomy was performed as soon as breathing became difficult. All four rabbits died in several hours. Sections of tissue from the tongue and the entire larynx were removed and fixed in 10 per cent formalin.

Hematoxylin-eosin and Giemsa stains were employed. The edema is not quite so striking after fixation as on the gross speci-

men but still is apparent, and in the larynx extends into the muscles. There was also a slight infiltration of eosinophil cells. Edema and eosinophil cells were absent in control specimens. The maxillary sinuses of the four experimental rabbits showed no edema or eosinophilic infiltration.

The combination of edema and eosinophilia gave a suggestion of an allergic mechanism. In this connection, it is of interest to note the findings at autopsy of the larynx in a case of fatal angio-neurotic edema of that region. Jackson¹ quotes Koenig, who found a marked infiltration of eosinophil cells and edema which spread into adjacent muscle tissue. Jackson assumes the mechanism involved is an increased permeability of the capillaries of this region. Since paraphenylenediamine edema and allergic reactions give a similar microscopic picture, the mechanism of allergy may be involved in irritant amino derivatives of protein to which the subject may be sensitive. The action of this protein derivative, or perhaps entire protein, is similar to a weak histamine-like reaction affecting the vessels of the head and neck, which are naturally more sensitive to these substances. In this way the location of edema of the larynx and upper respiratory tract can be accounted for.

In regard to the effect of various amines on all permeability, the work of Jacobs and Stewart⁶ is of interest. They found in a homologous series of amines, that the greater the length of the hydrocarbon portion of the molecule, the ease of entrance into the unfertilized eggs of the *arbia* (sea urchin) is correspondingly increased. The first member of the series formamid could not be used on account of its toxicity. However, on testing acetamid, proprionamid and butryamid, the permeability constant showed an increase in the order in which they are named. They demonstrated, in a quantitative manner, the qualitative principle stated by Overton,⁷ who stated that in a homologous series of compounds the ease of entrance into a living cell increases with the increase of length of the hydrocarbon portion of the molecule.

An immediate question to arise here would call for an explanation of the sudden edemas due to nonantigenic substances, such as aspirin, recurring in the larynx of certain individuals. The

recent work of Landsteiner and Scheer⁸ may throw light on this point. They proved that a crystalloid united with a protein carrier acts as an antigenic unit. They found that crystalloids in conjunction with protein injected into experimental animals caused the formation of an antiserum combining anticrystalline and antiprotein functions. This mechanism may explain the edemas of the larynx resulting from drugs in that they combine with some body protein after absorption from the intestine.

On a basis of these findings a tentative hypothesis for the formation of sudden edemas of the larynx may be formulated. The blood vessels of that region are more susceptible to the action of various amine compounds. In sensitive or allergic individuals the permeability of these blood vessels is increased by irritant complex amine compounds (protein and derivatives thereof) with resultant edema. Removal of the irritant automatically results in relief. The selective exact reason why blood vessels of one region of the body can be rendered more permeable than in others cannot as yet be given. It is hoped that this study will stimulate further investigation in this direction.

CONCLUSIONS.

1. Paraphenylenediamine injected subcutaneously produced marked edema of the mucous membrane and muscles of the larynx. Histologic picture shows eosinophilic infiltration.

2. The combined edema and eosinophilia suggests a resemblance to allergic manifestations, the mechanism of which is discussed in the text.

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LXVI.

THE SIGNIFICANCE OF EUSTACHIAN CURETTAGE.*

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Due to anatomic enlightenment, the proper interpretation of roentgenograms and the progress in histopathology of the temporal bone, we are in a better position to understand the reasons for recurrent suppurative otitis media and recurrent mastoiditis. In many instances they are due to infections situated in the remote recesses of the temporal bone.

Until recently it has been thought that recurrent suppurative otitis media and recurrent mastoiditis were caused mainly by middle ear infections, eustachian pathology and mastoiditis. Within the past year, there have been sporadic articles dealing with this subject along different lines. It has been pointed out that pneumatization is not limited to the mastoid region but is more widely distributed, and owing to a great variation in the pneumatization, distribution, size and location of the cells, they are therefore not limited to the mastoid cavity but may extend through various routes involving the eustachian tube, carotid canal and petrous pyramid.

Normally all mastoids become pneumatized. Should suppuration occur before complete pneumatization has taken place, there is a cessation of pneumatization (according to Wittmaack). However, there are instances where pneumatization continues irrespective of a suppuration. This may be due to the fact that the ingrowing mucous membrane was not completely affected, and it therefore did not check the progress of pneumatization. When a chronic suppuration is superimposed upon an already pneumatized mastoid, sclerosis, osteitis and bony eburnation take place in the path of the infection. The distant cells, such as the

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perilabyrinthine, peritubal, the petrous apex and carotid canal, may continue to remain infected and reinfected and therefore act as a source of infection, producing chronic suppuration or even recurrent mastoiditis. Every now and then a petrositis occurs in the pneumatized temporal bone. As previously described, this is due to the fact that the remote cells or the extramastoid cells become involved.

In an X-ray study of chronic suppurative otitis media, we were able to discern cellular areas in the deeper recesses of the temporal bone and we concluded that a chronic suppuration may originate from the following sources:

1. Any component part of the middle ear.
 - (a) Tympanic cavity.
 - (b) Eustachian tube.
 - (c) Mastoid.
 - (d) Epitympanic spaces.
2. Extension of the infection from the middle ear and the eustachian tube pressing inward from the tunica propria.¹
3. From the subarcuate area adjacent to the superior semicircular canals.
4. From the peritubal cells into the pyramidal tip (the perilabyrinthine route).
5. From the peritubal cells into the carotid canal, directly through eustachian dehiscences.²

Relative to the question of dehiscences, we recently made some anatomic studies at the laboratory at Temple University to determine whether dehiscences occur around the eustachian tube. We observed that many dehiscences occur in the cartilaginous and osseous portion of the eustachian tube. I might say that these findings coincide with those of Drs. Druss, Batson, Meltzer, McMahon and Kopetzky.

Dr. Joseph G. Druss of New York states that from a number of sections of the petrous pyramids dehiscences are not infrequent, particularly in infants and children, where they may be present in from 50 to 60 per cent of the specimens.

Dr. Oscar V. Batson of Philadelphia tells us that he is familiar with the dehiscences normally present in the cartilaginous part of

the tuba auditiva (Eustachii), as well as the gaps between the bony portion and the medial cranial fossa and between the bony portion and the structures of the base of the skull. He has no difficulty in demonstrating these breaks in the skeletal continuity in material obtained in the laboratory.

Dr. Philip E. Meltzer of Boston, in a survey of temporal bones, noted that fissures appear over the eustachian tube. On section of this region it was disclosed that the type of cell present is that which is closely related to the type of cell found around the mastoid and that frequently dehiscences do occur.

Having reviewed the possible avenues of infection, and assuming that all therapeutic measures have failed, the only other alternative is operative interference.

When a thorough radical mastoidectomy is performed we attempt to accomplish the following:

- (a) Exenteration of all necrotic cells.
- (b) Curettage and removal of all pathologic tissue in the middle ear (with the exception of the stapes).
- (c) Curettage of the eustachian tube.

We curette the eustachian tube so as to remove the pathologic mucous membrane and at the same time we invert it so as to convert the patulous eustachian tube into a closed cavity. This procedure is orthodox.

Because of anatomic enlightenment regarding the cellular distribution, and because we are aware of the presence of dehiscences in the eustachian tube, and also that there are cells present in the carotid canal and petrous portion of the temporal bone, we feel that the obliteration of the eustachian tube plays only a small part in what is accomplished by curettage of the tube.

SUMMARY.

Curettage of the eustachian tube accomplishes the following:

- (a) Breaks up the pathway of infection which often exists either by continuity or through the tunica propria.
- (b) Removes the diseased tubal mucous membrane.
- (c) Converts a patulous tube into a closed cavity.
- (d) Since it is an established fact that there are dehiscences in the eustachian tube and that the infection often takes place

through that route, then, it is possible that by curetting the tubal mucous membrane, drainage will be promoted from the surrounding and overlying cells through the dehiscence area. Hence curettage may promote drainage from the carotid canal and the deeper recesses through the dehiscence area.

1915 SPRUCE STREET.

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LXVII.

CALCIFICATION AND OSSIFICATION OF THE EXTERNAL EARS.*

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In a review of the literature on calcification and ossification of the external ears observers have reported forty cases which come under this classification. In the majority of the cases reported this finding was made accidentally during a routine examination of patients. A case is herein reported which was discovered similarly during the routine laryngeal examination preceding thyroidectomy.

K. C. H., white, female, aged 53, entered the hospital because of a prominent swelling in the neck. She had lost 45 pounds in the past two years, showed marked exophthalmos, nervous excitability, sweaty palms and abnormal appetite; all these symptoms had existed six or seven years.

The illness began six or seven years ago when the patient had a miscarriage (three months), at which time she was in a generally debilitated condition. Shortly after the miscarriage she noticed a prominence of both eyes, an enlargement of the neck, nervousness, sweaty palms, tachycardia and increased appetite. These symptoms and findings became progressively more pronounced until entrance into the hospital on November 11, 1930. About four or five years ago the patient noticed a very firm consistency of both auricles and that they were not mobile like normal auricles. The cartilage of both apparently became calcified and partial fixation to the skull was the result.

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Thesis submitted to the Faculty of Otolaryngology of the Graduate School of Medicine of the University of Pennsylvania in partial fulfillment of the requirements for the degree of Master of Medical Science (M. Sc. [Med.]) for graduate work in otolaryngology.

Family History.—The father died at 71 years of age of dropsy. The mother died of kidney trouble at 70 years of age. She had six brothers, four of whom had died in infancy, the cause unknown, and two sisters, one of whom had died in infancy, the other living and well.

Past History.—The patient had always been well and strong but easily excited and sensitive. She had had measles at the age of six, uncomplicated; influenza in 1918, uncomplicated. Aside from being nervous and excitable, she was well and healthy up to the time of the miscarriage, six or seven years previously.

Menstrual History.—Onset at age fourteen years. Regular, 28 4/5 days. Not painful. Menopause, one year ago.

Marital History.—One child, age 24, living and well. The patient had had two abortions, both criminal. She had had one miscarriage, six or seven years ago.

Systemic History.—The eyes showed bilateral exophthalmos with questionable diplopia. There was no pain or discharge from the ears and no tinnitus or vertigo. About four years ago the patient noticed that her auricles were very firm and that she was unable to move them about normally. There was no pain associated with these progressive changes. There was no nasal obstruction and no nasal or postnasal discharge. She had had no headaches. There was an enlargement of the neck in the thyroid area. No lymph glands were felt. The patient had considerable difficulty in turning her head to the right or left. There was some fixation of the neck. She had had no sore throats. The tonsils had not been removed. She had had no quinsy. She had had occasional attacks of hoarseness with colds. There was no difficulty in swallowing. All her teeth had been removed three years before, due to pyorrhea. She has plates at present. There was no dyspnea, no edema or precordial pain, and no history of colds. Her appetite had always been good with increased appetite the past three or four years. There was no nausea or vomiting. She had had hemorrhoids the past five years which had not bled. There was no constipation. She had had nocturia three times. There was some swelling of legs without pitting. She had had some yellowish discharge during menopause and has noticed it once or twice since.

Physical Examination.—The patient's temperature was normal, as was her respiration. Her pulse was 110. She was poorly nourished, co-operative and hyperesthetic. Her skin was warm and moist. The hair was graying and thin. The head was not notable externally. Brawny edema, tightness and lack of elasticity of the skin, especially over the abdomen, suggested scleroderma. Both auricles were bony hard. The auricular cartilage had entirely calcified and the auricles could not be moved. There was no tenderness. The hardening process extended inward, involving the fibrocartilage of the canal. The fibrocartilaginous canal was fixed to the bony canal. The fixation could not have been more marked had the bony canal been continuous with the calcified cartilage. This fixation of the canal wall had interfered with the normal outflow of cerumen and consequently in each ear there was impacted wax. The cerumen was more inspissated than normal, because it had probably remained in the canal for a long period of time. The tragus on each side was not only hardened but thickened. The remaining parts did not show any abnormal thickening. Both drums were retracted and many deposits of calcium were present in both. The whispered and spoken voices were normal, while bone conduction was increased somewhat in each ear. The Weber test showed no lateralization, and each ear showed a negative Rinne.

The audiometer showed that the hearing in both ears was reduced 10 to 15 units throughout the lower part of the range, with greater loss for high tones and a marked lowering of the upper limits for air and bone conduction. The negative Rinne in both ears, together with the marked lowering of the upper limit, suggested a combined conductive and perceptive lesion.

The eyes showed marked bilateral exophthalmos, 24 mm. in both eyes. There was marked widening of the palpebral fissures with definite lid-lag. The external ocular movements were normal. The pupils were round, regular, equal, and reacted readily to light but sluggishly to accommodation. Corneæ, lenses and mediæ were clear. The fundus examination of both eyes showed definite expansile pulsations of the arteries, including the second and third divisions as well as those immediately on and near the discs. They were expansile in contradistinction to transmitted

pulsations which are seen frequently in eyes because of the adjacent pulsating veins. This expansile pulsation was a phenomena in keeping with several pathologic conditions, one of which is the exophthalmos in thyrotoxicosis. The discs had a pale, waxy appearance, especially in the temporal borders. The fundi in general had a dirty pink reflex.

Roentgenographic Examinations.—Anterior posterior view of skull and mastoids. The film of the skull showed a rather marked degree of calcification in the auricular cartilages. This was also seen in the mastoid film. There was some thickening and increase in density in the triangular area of the right mastoid structure. The groove for the lateral sinus was also prominent. The opposite mastoid appeared clear. Diagnosis: Cartilaginous calcification of the auricles. Right mastoiditis.

Anterior posterior and lateral views of cervical spine: There was a moderate degree of hypertrophic osteo-arthritis, best visualized between the fourth and fifth cervical vertebrae. Diagnosis: Hypertrophic osteo-arthritis of cervical spine.

Basal Metabolism.—The basal metabolism test on entrance, November 13, 1930, showed plus 32. On November 20, 1930, following seven days of iodine administration, the basal metabolism had dropped to plus 10.

Other laboratory tests showed blood nonprotein-nitrogen, 28 mg.; blood sugar, 185 mg.; fasting blood sugar, 104 mg., and blood Kahn, negative. Blood calcium was 10.9, the normal being 9-11 mg. for each 100 cc., and blood phosphorus, 4.7 mg., the normal being 3.7 mg. for each 100 cc.

On November 28, 1930, the patient had a subtotal thyroidectomy. Her recovery from the operation was uneventful.

Gross pathology of thyroid removed: Material consisted of a nodular gland about twice the normal size. The capsule was present on all except the most posterior aspects of the two lateral lobes. The gland was soft, and on the cut section had a glistening appearance. There was a somewhat increased fibrous trabeculation of the glandular structure. Gross diagnosis: Toxic adenoma.

Microscopic pathology of thyroid removed: There were very few areas of hyperinvolution in the thyroid. The colloid, however, for the most part took a fairly good stain. The cells were columnar and low columnar, and here and there presented plication into the lumen of the acinus and hyperplasia. Fibrosis and lymphocytic infiltration were present but not prominent. This represents the picture seen in a moderately good response on the part of the thyroid to iodine therapy. Diagnosis: Exophthalmic goiter . . . iodine therapy.

Histologic Examination of the Tissue Removed From the Auricle.—Serial sections were made, parallel to the flat surface of the biopsy, whose dimensions were $\frac{3}{4}$ by $\frac{1}{4}$ cm. Under the microscope the sections showed evidence of cracking on removal.

The upper and lower superficial surfaces showed compact lamellar bone with only a slight suggestion of cartilage cells. (Fig. 1.) Some areas showed bluish stained necrotic regions. A few bone marrow spaces, mostly filled with fat cells, were seen scattered throughout the lamellar bone. (Fig. 2.)

In the deeper layer of the ossified auricle or in the middle of the piece from the biopsy, marrow spaces occupied almost the whole area. (Fig. 4.) Red blood cells could be seen in them, young vessels coursing across the fat cell, patches of large mononuclears, polymorphonuclears and small mononuclears. Red blood cells in places were diffusely scattered among the fat cells; in places they laid in rouleaux. Throughout the marrow spaces in the deeper part of the tissue were areas of lamellar bone separated more or less by the marrow spaces and fat cells. (Fig. 3.)

There was no alignment of osteoblasts indicating bone growth in activity. Very few large mononucleated cells were in evidence (osteoblasts).

No evidence of fibrous connective tissue in scar tissue formation was seen in any of the sections.

Histologic Examination of Normal Auricular Cartilage.—A comparison of the above findings with histologic examination from auricular cartilage of an infant two days old, as seen in the microscopic section, was as follows:

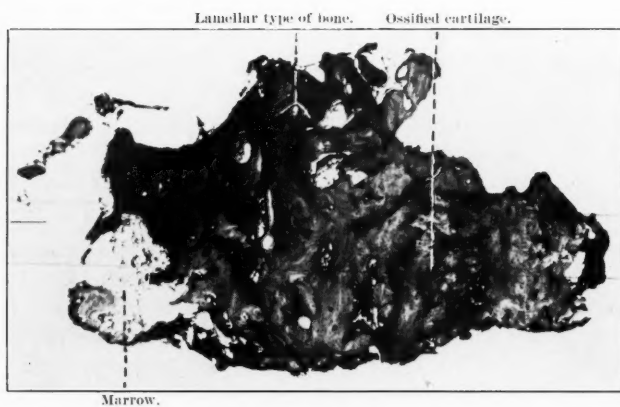


Fig. 1. Showing almost solid bone in the more superficial layer of the ossified auricle.

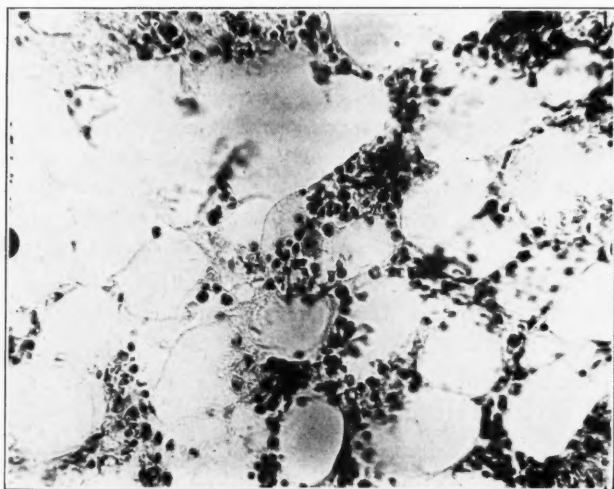


Fig. 2. Detail of one of the few marrow spaces seen in Fig. 1.

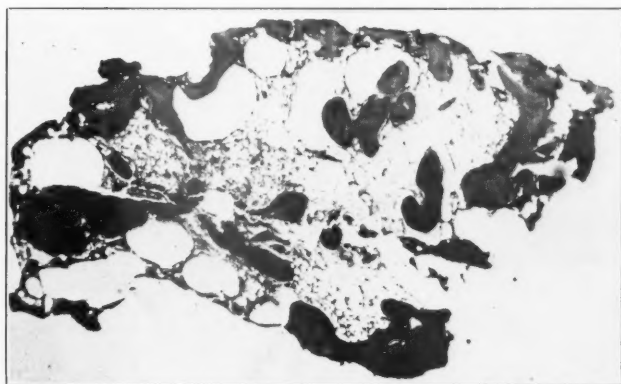


Fig. 3. The cancellous type of bone found in the deeper layer of ossified auricle.

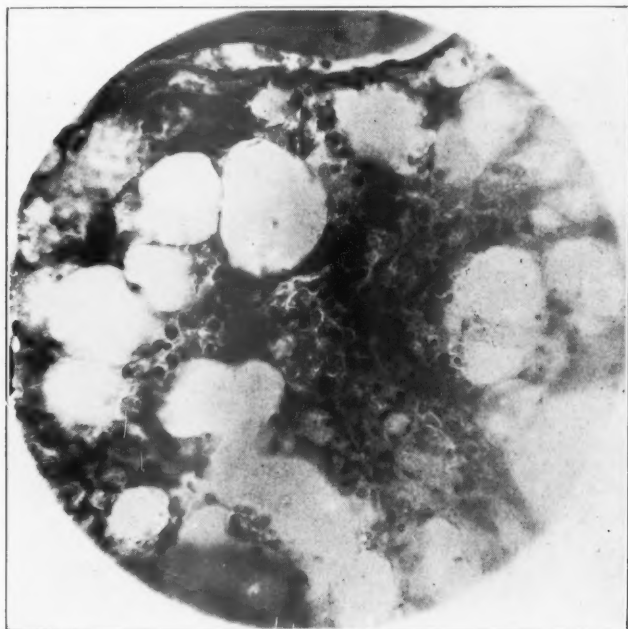


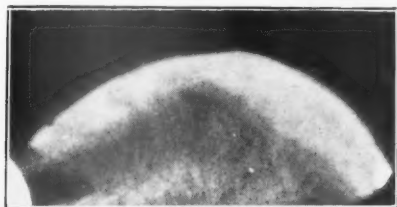
Fig. 4. Detail of one of the many marrow spaces seen in Fig. 3.

The auricular cartilage is composed of yellow elastic fibrocartilage. The majority of the fibers run in more or less parallel rows at right angles to the long axis of the pinna. The cartilage cells are encapsulated either singly or in pairs. These cells lie with their long axis directed at right angles to the long axis of the pinna. As Eckert-Möbius¹ has stated, these cells shift their direction toward the perichondrium and in that region lie parallel to it. The perichondrium appears to be very thick—eight or ten rows of nuclei may be counted. The adjacent muscles show well marked striations. Subcutaneous fat, sebaceous gland, hair follicles and epidermis flank either side of the cartilage.

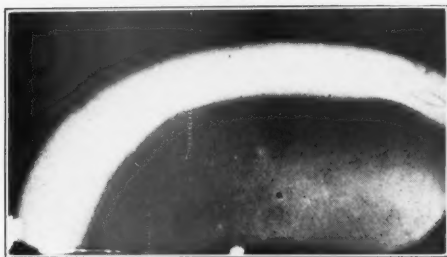
Roentgenographic Examination of Ossified Auricles.—Roentgenograms of ossified auricles made with dental film placed behind the auricle and the tube laterally. Such examination shows a marked increase of density of the auricle, indicating material calcium content. There is also a faintly reticulated structure such as that noted in the roentgenograms of cancellous bone. (Figs. 5 and 6.)

Routine Physical Examination of Auricles for Areas of Calcification or Ossification.—Over 800 pairs of auricles were palpated during routine ear, nose and throat examinations in the Washington University Out-Patient Clinic, the ages of the patients varying from fifteen to seventy-five years of age. In no instance was there any suggestion of hardness or immobility. As no areas were felt which might be said to be calcified or ossified, it was not possible to take roentgenograms of any suspicious auricles for verification by roentgenographic examination. Therefore, ten cases were selected at random. The ages of these persons were approximately that of the patient in whom we are interested.

Comparison of Roentgenograms of Ossified and Normal Auricles.—These were comparison films made on the auricles of ten normal persons. In these roentgenograms the images of the auricles were a homogeneous nonreticulated and much less dense structure than in the case of the patient with the ossification. (Figs. 5 and 6.) The examination in this instance indicated an extensive ossification of the auricles.

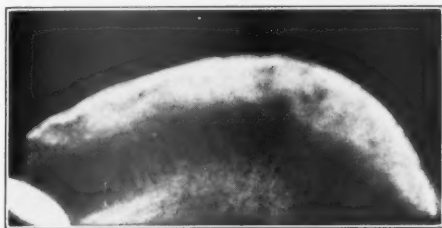


Ossified ear cartilage (right).

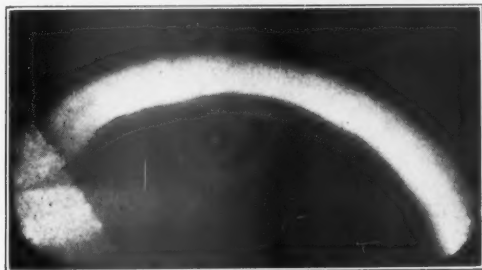


Normal ear cartilage (right).

Fig. 5. Roentgenograms of ossified and normal auricular cartilages (right).



Ossified ear cartilage (left).



Normal ear cartilage (left).

Fig. 6. Roentgenograms of ossified and normal auricular cartilages (left).

Chemical Examination of Biopsy From One of the Ossified Auricles.—Chemical examination and analysis of the bone from the auricle showed it to have 21.59 per cent total calcium and 9.02 per cent phosphorus in the dried specimen. The ratio of total calcium to total phosphorus in this specimen is 2.39.

Normal bone, according to F. H. McCrudden,² is composed of calcium oxid, 28.85 per cent; magnesium oxid, 0.14 per cent; phosphorus P_2O_5 , 19.55 per cent; and sulphur, 0.14 per cent, which when changed to the elements are calcium, 20.6 per cent; magnesium, 0.085 per cent; phosphorus, 8.73 per cent, and sulphur, 0.14 per cent.

The ratio of the total calcium to the total phosphorus in the normal bone, according to McCrudden, would be 2.36. A comparison of the ratio of the total calcium to total phosphorus in the abnormal bone from the auricle and normal bone, namely, 2.39 and 2.36, seems to show that the new bone formation in the auricles is true bone.

Peters and Van Slyke³ reported the ratio of total calcium to total phosphorus in normal bones varying from 2.1 to 2.2, which again compares favorably with the calcium phosphorus ratio obtained from the biopsy.

Howland, Marriott and Kramer⁴ studied the ratio of the calcium to phosphorus in many infant bones, and their ratios varied from 2.15 to 2.38. In their data they expressed their ratio as of residual calcium over phosphorus, but, since the data which I used above in the abnormal ear bone considered only total calcium, I changed their ratios so that the result would be expressed in total calcium over total phosphorus, in order to use them as a means of comparison with our results.

Kramer and Shear⁵ reported this ratio of total calcium to total phosphorus in normal bones varying from 2.11 to 2.39.

In their article they had data on some abnormal calcification of tissues, such as thyroid gland calcification and tuberculous lymph glands. These gave ratios varying from 2.22 to 2.38.

They also had three cases of calcified fibroids in which the ratio of total calcium to total phosphorus was abnormally high—i. e., 2.6, 2.54 and 2.53. Again, the above ratios were originally represented as residual calcium over phosphorus, but for means of

comparison I changed the ratios to represent total calcium over phosphorus.

Etiology of Cartilage Calcification and Ossification of the Auricles.—The etiology of calcification and ossification of the auricular cartilage has been attributed to numerous causes:

(1) Unusual spontaneous changes occurring in senility with poor nutriment of peripheral parts of the body.⁶ Frankel,⁷ in 1919, expressed this view of calcification and ossification. In Bailey's⁸ textbook, he says: "With old age, cartilage loses its translucency and bluish-white color and appears yellowish and cloudy. This change is due to the practical loss of chondromucoid and the large increase in the amount of albumoid. Calcification is also of common occurrence in old cartilage, and is usually associated with degenerative changes of the cartilage cell. The calcareous particles are at first deposited in the vicinity of the cells and then slowly spread into the matrix.

D. P. Barr,⁹ in 1930, stated that calcium is deposited wherever caseation, necrosis or hyalinization was seen, or where the vitality of the tissues is diminished by imperfect blood supply. In scleroderma and Raynaud's disease we see abnormal calcification of the skin and subcutaneous tissues more often in the extremities and in parts in which the circulation is obviously impaired.

(2) Freezing and frost-bite have been reported by many as the cause of calcification and ossification. Chief among those so reporting are: Linsmeyer,¹⁰ 1889; Wassmund,¹¹ 1889; Lübbers,¹² 1912, three cases; Frankel, 1919, two cases; Haugh,¹³ 1906; Müller,¹⁴ 1921, two cases; and Martenstein,¹⁵ in 1926.

(3) General debilitating diseases were given as the etiology in a case reported by Laskiewicz,¹⁶ in 1924, in which there was partial bilateral ossification of the auricles occurring during the course of severe diabetes.

(4) Trauma and perichondritis¹⁷ was given as the cause by several authors: Frankel, in 1919, reported three cases following puncturing of the ears. Gray,¹⁸ in 1925, told of a case following hematoma of the auricle. Gudden,¹⁹ in 1889, mentioned a patient with ossification of the auricle following insect bites, and Knapp,²⁰ in 1892, told of one following a prolonged perichondritis of the auricular cartilage.

(5) Endocrine disturbance may be a possible cause of calcification as in the case herein reported. The patient had marked pathologic changes in the thyroid gland with no other known cause.

(6) Syphilitic perichondritis or perivascular infiltration was given by Ekehorn,²¹ in 1925, as the most probable cause in a reported case of ossification of the auricle in which the patient was strongly suspected of having hereditary syphilis.

(7) Inherent properties of ossification of cartilage which lie dormant similar to malignant tumors was given as a possible cause by Marobbio,²² in 1928, in a case report in which no other cause could be found to account for the calcification and ossification of the auricles.

From a study of the above cases, the outstanding factors in the ossification and calcification of the auricular cartilage are malnutrition, severe local inflammation, or some disturbance in the circulation of the part, as exposure to intense cold.

CASES OF CALCIFICATION, OSSIFICATION, OR BOTH, TO DATE.

1866—Bochdalek²³ reported a case of calcification of the auricular cartilage. It was found by accident in the dissecting room in a man, 65 to 70 years old. The left helix and part of the right helix were calcified. No other pathology was found in the skin or other parts of the body. The calcification on microscopic examination was true bone.

1869—Votolini.²⁴ Patient in whom there was ossification of the entire posterior surface of the auricular cartilage on both sides. The age of this patient was 70 years.

1870—Gudden. Case report of a man, 70 years old, in which there was complete ossification of the auricular cartilage, the cause of which was unknown.

1885—Schwabach.²⁵ Reported a case with symmetrical ossification of the auricular cartilage. The ossification was localized to the upper and posterior part of the helix and antihelix. The patient was 55 years of age, and this calcification of the auricular cartilage existed since childhood.

1889—Gudden. Unilateral ossification of the auricular cartilage in a young man, in which the probable cause was chronic

inflammation from insect bites, seven years previously, causing nodules and cysts in the auricle. The auricle was small but thickened, with irregular surfaces. One of these irregularities was removed and it proved to be true bone with haversian canals.

1889—Linsmeyer reported a case of a 75-year-old man with ossification of both auricular cartilages. In this case there was only partial ossification in the helix and antihelix of both sides. The patient consulted Linsmeyer on account of pain in the ears from pressure upon them when reclining. This patient gave a history of having had his ears frozen when a young man.

1892—Knapp told of a young man, age 24, with history of injury to one ear followed by perichondritis. The ear showed partial ossification in the region of the helix and antihelix.

1895—Osler.²⁶ Three cases were seen within six months with calcification of part of the auricle of one ear without presence of trophi, without arthritis, and in each instance without any history of acute gout.

1899—Wassmund reported a patient, age 49, with marked induration of the right auricle following freezing. Wassmund examined the auricle by roentgenograms and true bone formation was found. The ossification was found in the helix and scaphoid area.

1906—Haugh wrote of a man, age 64, who gave a history of having had the left auricle frozen 32 years previously. After the time of freezing he consulted numerous physicians for impairment of hearing in the left ear, which was relieved each time by removing a large plug of cerumen from the canal. The left auricle was rigid and hard to the touch. The ossification involved the helix, scaphoid area, fossa triangularis, antihelix, part of the concha and the antitragus. The lobule was of normal consistency. The skin over the auricle had a slightly yellow color but otherwise was normal.

1912—Lübberts made a histologic and roentgenographic study of five cases of ossification of the auricles. All five cases showed true ossification of cartilage and formation of spongy and fibrous tissue. The ossification in these cases occurred mainly in a localized area in the region of the helix and the scaphoid. The ages of

these five cases were all over 45 years. The etiology in three of the cases was freezing of the auricles. In one case the cause was unknown, with no history of any trauma. The fifth case was found at autopsy and the cause could not be discovered.

1919—Frankel. Up until 1919 all the cases reported spoke of the ossification of the auricular cartilage as being a rare condition, but Frankel thought differently, for he noticed that in people of advanced years there were small, and occasionally large, circumscribed areas of the cartilage which were hard and rigid. He then made roentgenograms of the ears of 75 patients, 52 men and 23 women, between the ages of 45 and 89 years. Of these, 15 showed definite areas of calcification and ossification of the cartilage. Thirteen were men and two were women. In ten of these fifteen cases the areas of calcification and ossification were no larger than 2 mm. in diameter, and were symmetrical and equal on both sides. In the majority of the cases the pathologic changes occurred in the helix and antihelix. Of the fifteen cases, nine showed calcification, three showed ossification, and the last three showed both calcification and ossification.

1921—Müller told of two cases he saw in his clinic, in which the auricles were hard and rigid. He thought at the time that these changes were probably caused by exposure of the ears to cold and freezing, either early or late in life, and that they usually occurred in older people.

1924—Laskiewicz reported a case of a man, aged 49, in which the upper half of the auricle had become hard and rigid following a severe attack of diabetes. In this case there was no history of freezing or trauma to the ears.

1925—Gray. Higbee, in his paper, told of a case reported by Gray, of bilateral calcification of the auricular cartilage. The probable etiology was injury to the ears twenty years previously, in which hematomas were formed in the auricles.

1925—Ekehorn gave a case history of complete calcification and ossification of the cartilages of both auricles. In this patient, age 42 years, the auricles, except for the lobules, were bony hard. Since ten years of age, he had had certain diffuse noncharacteristic skin sensations in both legs which became worse. In later

years he began to have pains localized in the muscles and the bony shafts of the legs which were worse at night. These were not lancinating pains. The paresthesia was like that occurring in syphilis, otherwise there was no peripheral involvement or central localization. The pains in the legs were thought to be caused by syphilis, because they disappeared after antisyphilitic treatment. At the age of fourteen he also had had ulcers of the skin over both tibias. These lesions began as pimples and progressed to ulcerations. He was confined to his bed for two weeks, with no fever or local pain. Following the oral administration of mercury the ulcers disappeared. The resulting scars were not typical of syphilis or characteristic of any other disease. The localization was typical of syphilis and he probably had had periostitis of both tibias. The patient had a dilatation of the aorta and a distinct increase of the pulse on the left side. He also had had chorioiditis and parenchymatous keratitis in childhood. The history of hereditary syphilis was considered. In childhood, from four to five and from ten to twelve years of age, he had had bilateral otitis media, about which very little was known. At age twenty both ears were treated for discharge by irrigation. The hearing was reduced in both ears and varied with head colds. At this time, when he was twenty years old, the patient noticed a firmness of the auricles. This change came on so slowly that it was impossible to determine any association with the otitis media. The auricles, except for the lobules, were bony hard. They felt irregular and the most pronounced changes were in the tragus and the antitragus. They had a normal external picture and there was no evidence of trauma. There was no history of freezing of the ears, no history of trauma, of perichondritis or of piercing of the ears.

In March, 1925, he was in the hospital for a hand injury, at which time the Wassermann and spinal fluid tests were negative.

Thyroid and testes were normal. Blood pressure was 140/100. The lungs showed hyper-resonance, rough breathing with sonorous râles.

The nervous system was negative.

The blood vessels and the heart were normal. Cella turcica and hypophysis were normal.

The larynx and cartilages of ribs showed no calcification. The right knee showed no changes and the position of cartilage was normal. There was no calcification there.

At this time roentgenographic examination of the auricles showed the cartilage of both infiltrated with calcium, and a definite calcification of the antihelix and antitragus. The roentgenograms showed cloudy areas in places and a network of spongy-like structure. Here and there were seen small clefts which had the appearance of fracture lines.

There was little known about the family history except that the father had a blood disease.

1926—Martenstein reported a case of a man, about 63 years of age, with a history of having had the ears frozen at about twenty years of age. Both auricles showed ossification with hardening of the auricles.

1928—Marobbio reported a case of a woman, aged 58, with no history of freezing of the ears, trauma or piercing of the ears, who had always been in good health. Areas of ossification in the cartilage of both auricles in the region of the antihelix were found. No known cause or time of onset could be given by the patient. A roentgenographic study of her auricles showed true bone structure.

1931—Higbee²⁷ reported a case of a man, aged 82, with history of impacted cerumen in the ears. The left ear was somewhat larger than the right. It protruded more prominently and had a slightly bluish-gray color. The ear, except for the lobule, moved en masse and was bony hard. The ear drums and hearing were normal. The condition of the left ear had been present for about six years, and the patient could give no cause for the trouble. The records showed normal findings in regard to blood cells, blood pressure, chemical content of the blood, Wassermann test and function of the kidneys and the heart. A roentgenogram revealed a large area of calcification, 3 cm. in diameter, in the region of the antihelix and the helix. A second area of calcification paralleled the first one. In these calcified areas the periphery was more dense than the center, and a reticular structure was seen similar to the formation of spongy bone.

SUMMARY.

1. Calcification and ossification of the auricles of the ears is a comparatively rare finding in ear pathology.

2. One additional case is herein added to the forty cases which have been reported by nineteen observers during the past sixty-five years

3. The age incidence is from 22 to 89 years, and the majority of the cases reported were more than 53 years of age.

4. The cause has been attributed to numerous factors, namely, unusual spontaneous changes occurring in senility with poor circulation of the peripheral parts of the body; freezing and frost bite; general debilitating diseases; trauma and perichondritis; syphilitic perichondritis or perivascular infiltration; and abnormal inherent properties of ossification of cartilage. On the basis of the findings in the case herein reported endocrine disturbance may be added as another possible cause.

5. Small and sometimes large areas of the auricular cartilage become ossified and can be felt on palpation. In the case reported there was complete ossification of the cartilages. The entire auricles, except for the lobules, were bony hard. There was a complete fixation of both ears to the skull with complete immobility.

6. The routine physical examination of 500 pairs of auricles of the ears failed to reveal any evidence of hardening nor were any areas felt which might be said to be calcified or ossified.

7. Roentgenographic examination of the ossified auricles showed a marked increase in the density of the auricles, indicating material calcium content. There was also a faintly reticulated structure such as that noted in roentgenograms of cancellous bone.

8. The routine roentgenographic examination of the auricles of ten elderly persons showed no areas of calcification or ossification.

9. A chemical analysis of the biopsy taken from the ossified auricle showed a calcium phosphorus ratio approximately that of true bone.

10. The histopathologic examination of the tissue showed a compact lamellar bone with only a slight suggestion of remaining

cartilage. Throughout the lamellar bone there were marrow spaces filled mostly with fat cells.

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LXVIII.

TERATOMA OF THE ANTRUM IN THE NEWBORN.

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Although there have been many cases cited in the literature of the finding of teratomata in the mouth and other places in the body, I have been unable to find another case where the growth originated in the antrum of the newborn. Through the kind assistance of the American College of Surgeons, a very careful check of the medical literature was made.

Family History.—Mother living and well, age 26; Wassermann negative. Father living and well, age 33; Wassermann negative. Sisters living and well, ages 3 to 5. Brothers living and well (1), age 7.

Maternal Grandparents.—Grandmother living and well, age 45. Grandfather dead, age 37; cause unknown.

Paternal Grandparents.—Grandmother living and well, age 48. Grandfather living and well, age 50.

Personal History.—Six hours after delivery by a midwife a female colored baby was admitted to the Natchez Charity Hospital at 12:45 p. m, by Dr. Wallin of the health unit, on account of a large tumor mass protruding from the oral cavity, causing difficult respiration. The delivery had been normal in every respect, and performed by a negro midwife.

Physical Examination.—Reveals a newborn negro infant, head normal shape and contour; no scars; ears negative, eyes negative, nose negative. On the right side of the face there is a uniform swelling extending from the infraorbital region to the margin of the maxilla. The zygoma is pushed outward in a perfect semi-circle. The swelling is firm to the touch and does not fluctuate. Examination of the mouth shows a large, red, pedunculated mass protruding from the oral cavity. The right half of the hard palate is absent, the space being occupied by the growth. The antral cavity is greatly enlarged with no definite bony walls, and the

cavity is filled with the tumor mass. The tumor also filled the mouth, making respiration difficult and nursing impossible. The left side of the face is normal.

Examination of the heart and lungs reveals no pathology.

Abdomen soft; no palpable tenderness or tumor masses. The umbilical cord is normal in appearance.

The extremities are normal.

The skin shows no rashes or other pathology.

Photographs Nos. 1 and 2 are inserted here to show the points in the physical examination so far along with a comparison of the size of the tumor to the size of the head.

Course in Hospital.—The patient was admitted and morphin sulphate, grains 1/72 was given. Patient was sent to the operating room for possible removal of the mass. Dr. J. S. Ullman removed the portion which extended outside the mouth by ligation and electrocoagulation in the region of the right alveolar process. The macroscopic findings at that time were as follows: "Irregular cystic mass protruding from the mouth about 10 cm. in diameter and extending up to the right cheek. The tongue is not involved." The

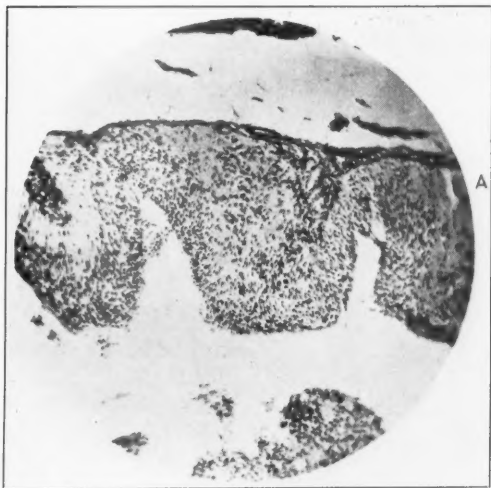


microscopic findings, made by Dr. John Langford, are as follows:

"Grossly the specimen is a dark reddish black mass, approximately 10 by 8 cm., rather soft and fluctuant in consistency, apparently made up of a number of cavities. On sectioning there are noted many multilocular cavities filled with blood or bloody material, and the tissues between the cavities are for the most part rather soft, there being no evidence of bony structure. Histologic study shows a number of cavities, some of which are lined with endothelial cells, others stratified squamous or columnar epithelium. Between these cavities are a great number of dilated blood capillaries and groups of cells which are arranging themselves



in a variety of forms. Some are apparently attempting to form lung tissue, lymph nodes and nerve tissue. One relatively solid mass gives the histology of convolutions of the brain, and among the nerve tissue in one area is a suggestion of an effort to form a cavity lined with ependymal cells. A variety of glandular structures are noted, some lined with ciliated epithelium, others with tall or low columnar and still others showing a papillary ingrowth. There is an aggregation of glandular structures which in their arrangement and histology suggest pancreas. The supportive structure is made up largely of connective tissue and smooth muscle tissue. No bone or cartilage is demonstrated.



A. Showing nerve tissue in form of convolutions of the brain.



A. Glandular structure of salivary gland. B. Blood vessel. C. Cavity lined with stratified columnar epithelium, probably of the respiratory system.

The histology is that of a teratoma, or a structure springing from a toti-potential cell which in its development has produced tissues representative of the several primary layers."

The operation was begun at 2:45 p. m., and completed at 2:56 p. m. The child was returned to bed. Adrenalin, minims two, were given by hypodermic injection and hot water bottles were placed around the body. The patient began to show signs of weakening and expired at 6:30 p. m.

The autopsy report was as follows: Name, Baby Sims; age, 12 hours; sex, female; race, colored. Date of death, April 30, 1931, at 6:30 p. m. Date of autopsy, April 31, 1931, at 7:30 p. m.

Autopsy Findings.—A well developed negro newborn female, apparently normal, with the exception of a mass involving the right antrum, infraorbital region, extending from the lateral wall of the antrum to the midline of the hard palate and filling the mouth. A fresh scar is seen, due to surgical removal of a portion of the tumor. The mass remaining in about the size of a hen's egg, arising in the antrum and right malar bone, replacing the right half of the hard palate and filling the oral cavity. The mass is of friable consistency and bleeds freely. The other organs of the body show no abnormal pathology.

Microscopic diagnosis: Benign tumor, possibly teratoma.

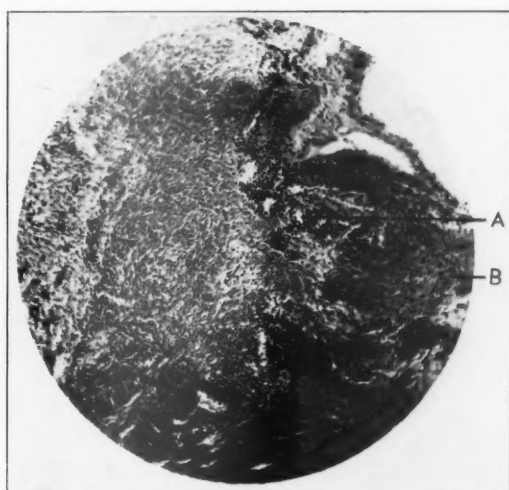
In my earnest effort to show the rarity of this type of case I have incorporated in my review a few cases of teratomata located in the vicinity of the antrum in the adult as well as the newborn. The different pathologic reports tend to show how different the structure appears under the microscope.

Stauffer, F., in the *Eye, Ear, Nose and Throat* monthly, reports a case of "nasal teratoma" in a woman, about 41 years of age. The abstract of this case is as follows:

"In the present case (woman, probably about 41), posterior rhinoscopy revealed an ovoid, smooth, pinkish tumor mass filling the left half of the upper nasopharynx. Attempts to remove it through the nose with a snare, or through the mouth with forceps, had to be abandoned on account of profuse bleeding. Electro-coagulation was then tried; the tumor was finally removed with a snare. Microscopically, it was invested with typical skin, later



Very low magnification, showing a number of cavities, most of which are lined with columnar epithelium—the tissue between is a mixture of glandular structures, brain, muscle and connective tissue. No bone or cartilage demonstrated.



A heterogeneous mixture of glandular structures and supportive structures, some of which is nervous in type. A. Glandular structures. B. Nerve tissue.

exhibiting surface keratinization and the specialized structures, viz., hair follicles, sebaceous and oil glands; internally it consisted of mucoid and more mature fibrous connective tissue background, studded with nerves, cartilage, smooth and striped muscle cells. Diagnosis: Teratoma.

"This form of growth is rarely found in the nose. Ballenger's 1925 edition has the following to say about teratoma. Quoting Lennox Brown: 'Teratomata are usually congenital and consist of tissue growth springing from two or three embryologic layers. They appear, therefore, most frequently in those regions where the various germinal layers are in close apposition. The pharynx, which develops from the junction of the neural and dermal epiblasts with the hypoblasts of the foregut, is, therefore, a suitable location for the growth of teratomata.'

"These growths are, therefore, always congenital and of slow development, as instanced in the present case. Here the symptoms (nasal obstruction, attendant secretion and progressive deafness) had existed for about six years; but no doubt this growth had attained considerable size many years prior to that time."

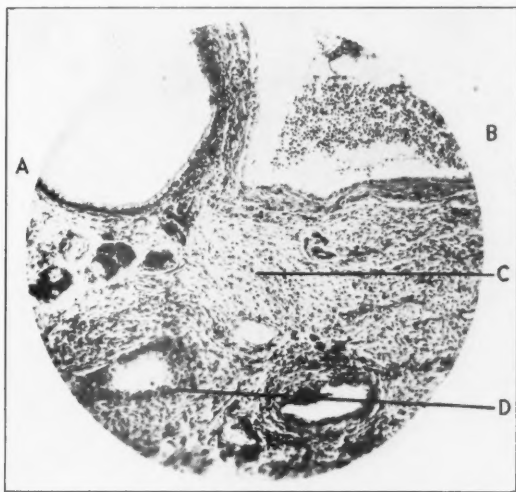
I would like to mention at this point that in my case, as you can easily note in the pictures, the growth exceeds in size at birth many times the size of the growth in the above abstract. You will also note from the pathologic report that even the pathology differs greatly.

Another case is reported by Bosch, titled "Teratoma of the Oral Cavity in a Two Months Old Infant." This report is abstracted from the *Rev. de med. y chirurg.*, 112, 453, 1916:

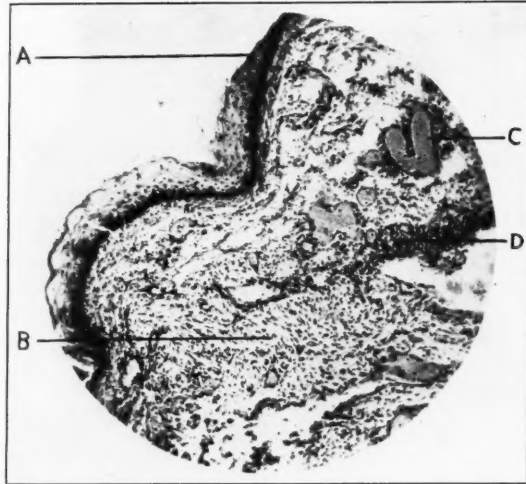
"The first signs of the affection were crying, guttural rhonchi, especially at night, poor sleep and the state of nutrition. The affection had been diagnosed by two specialists as retropharyngeal abscess. Examination revealed that the tumor consisted of an ovoidal mass with a smooth surface, the upper pole of which was hidden behind the palate, while the lower pole was hidden behind the root of the tongue. The color was a pale yellow. It was fairly indurated and could be displaced except vertically. A puncture of the tumor produced an abundant flow of blood. The tumor was cauterized. The tumor was of an ovoidal form and measured $2\frac{1}{2}$ by $1\frac{1}{2}$ cm. The pedicle was 7 mm. The sur-



Two large irregular groups of racemose glands above which is a dilated blood channel and an epithelial lined structure filled with blood and secretion—further upwards is the wall of a cavity lined with stratified epithelium.



Part of a dilated cavity lined with mucous forming (goblet) epithelium, separated by a thin supportive band from a large blood vessel containing usual blood cells—the structure beneath contains small gland groups surrounded by a loose network stroma in which nerve fibers are seen. A. Goblet lined cavity. B. Cavity filled with blood cells. C. Nerve tissue. D. Glandular structures.



A structure covered with stratified squamous epithelium resting on a loose vascular and edematous meshwork composed of nerve tissue and connective tissue. A. Stratified squamous epithelium. B. Nerve tissue. C. Blood vessel (vein?). D. Glandular structure.

face presented the characteristics of young normal epidermis covered with light hair. The histologic examination of the different layers revealed the presence of muscular striated, dermo-epidermic tissue, adipose, connective and embryonal tissues.

"Dr. Vidal believed that the rapid growth was due to rich vascularization. Dr. Clotet thought that it could be attributed to an evagination of the somatopleure. The regional structure showed a certain degree of predisposition toward that."

In a personal communication from Dr. Julian Chisolm of Baltimore he states that after making some inquiries and after looking over the history room of the Johns Hopkins Hospital he was unable to find any mention of such a case. He does say that in the index of the Pathological Building he found one case listed in a girl of 12, who died in October, 1926. In this case, he says the tumor had eroded the sella turcica and replaced the hypophysis, surrounded the oculomotor and trochlear nerves to both eyes and invaded the third ventricle. The point of origin was

thought to be the hypophyseal duct, although in the pathologic report it was noted that the cyst lining closely resembled the lining of the sphenoid and ethmoid cavities.

In another personal communication from Dr. Joseph C. Bloodgood, he states that he has never seen a teratoma of the antrum in the newborn.

From a general treatise on "Dermoid Cysts of the Antrum of Highmore," written in 1888 by Herman Doerner in Strassbourg, I would like to quote what he says on the subject of the time of appearance of this type of tumor: "Dermoids are usually discovered in young individuals. If they are not noted until later life it can be proved in every case that they have been in existence for a time. At the time of puberty all dermoids show particular development. Up to that time they may be small and scarcely noticeable; they suddenly begin to grow and cause disturbance. Premature development may be induced by trauma. Otherwise the course of dermoids is very slow and they may exist for a long time without acquiring any noteworthy size."

I feel that it would be useless to report any more of these articles, since there are none which have a direct bearing on this case, either as regards size, pathology or time of origin. I will, therefore, close this report with a few conclusions:

CONCLUSIONS.

1. That teratomata, while rather common in other parts of the body, are rare when the site of origin is in the antrum.
2. That no case in the literature has been found of a similar case in the newborn.
3. That in this case the development in utero was extremely rapid.
4. That the majority of teratomata are more nearly true dermoids in which can be noted the derivatives of the primary layers, i. e., teeth, hair, nails and even bony structures.
5. That in this case, from the pictures submitted, it is unique in size, pathology, site of origin and time of origin.

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LXIX.

THE TREATMENT OF CARCINOMA OF THE LARYNX.*

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SAN FRANCISCO.

In no place in the body, in the treatment of cancer, have we a problem to face such as is present in the larynx. Here alone we see cancer starting in a box which is comparatively shut off from the surrounding tissue by a strong cartilaginous wall, the invasion of which is slow. Here, par excellence, we can watch its development under the eye by means of our mirror and control its treatment according to the progress of the case.

It is, in this location, as protean a disease, however, as in any other portion of the body, requiring just as prompt and drastic measures to eradicate it as elsewhere. The presence of the box has in the past proven to be as much of a menace to the patient as a benefit in that it has had the tendency to lull the unwary physician into a comatose condition until it was too late for the surgeon to interfere. The literature is full of warning to attend to hoarseness early, yet I fear that the average practitioner does not read the literature and therefore does not get the signals. It is becoming better, however, and we are getting a larger and larger number of early cancers to study, thus helping in clarifying a very dark and stormy track.

Cancer here follows that of all other portions of the body in its tendency to a rapid or slow malignancy, some extending their life cycle to a few months, others to twenty years or more. Here, also, they exhibit the same tendency to build rapidly into a tumor mass filling the box, or on the other hand, remain flat on the wall, invading this and extending insidiously into the surrounding or distant tissues. The presence of the box is a help in the ultimate treatment, but doubly so if the surgeon can see the case before

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the invasion of the wall so he may have a chance to study the type of growth and its rapidity of action.

The form of treatment naturally depends first upon the age of the patient; secondly, upon the position of the growth in the larynx; thirdly, upon the extent of invasion; fourthly, on the social and economic status of the patient.

Age.—In youth we see carcinomatous changes taking place in papillary growths which have been present for years but which have caused but few difficulties beyond a chronic hoarseness. The carcinomata may be removed entirely by the removal of the papillomata from the cords or walls of the larynx by the indirect method of operating, thus saving the patient a capital operation and danger to his life through a laryngofissure.

Case one of my series is such a case; a boy of twenty-two who had had papillomata on the left cord all his life. These were removed in two sittings, under cocain, the cords remaining free of growth to date, a period of one year. Carcinoma, however, is not a disease of youth but of middle and late life and these cases are rare.

As we approach late life (eighty years of age), we find there is a tendency of larynx carcinoma to become less malignant than in middle life and we again have a chance to delay operative measures or treat the case expectantly, as they seldom build tumor masses which interfere with breathing. These cases appear like keratotic overgrowths, flattened on the sides of the larynx and showing no tendency to invasion of the surrounding soft tissues. It is in the carcinomata of middle life, from forty to sixty-five years of age, that our whole fund of clinical and surgical experience is drawn upon to choose that method of treatment which will best fit the case and bring it to a satisfactory conclusion. Here anything may occur, and the surgeon must stand ready to adapt himself to the emergency, changing his line of treatment to fit the change in conditions.

Position of Growth in Larynx.—The most favorable position for a successful removal of early cancer is the middle third of the cord. The cord may be removed in toto and a good, clean, functioning larynx remain, with a clear, loud voice. This may be

done by direct or indirect method by means of a knife or double curette, or by laryngofissure, but I believe that in all such cases there should be a fulgeration of the base and subsequent application of radium. Should this prove unsuccessful, a hemilaryngectomy or laryngectomy may be performed.

One of my cases handled by indirect method was a lawyer, who conducted his work for a year and a half with a clear voice, later dying of pneumonia. Another is evidently clear of his lesion, though the voice is extremely husky.

Those growths which occur on the arytenoids are, as a rule, very malignant, but one of my cases on the esophageal side of the arytenoid was easily removed together with a large portion of mucous membrane and has remained clean nearly a year.

Epiglottic growths are slow to grow, an amputation of the epiglottis being easily done if the growth is limited in extent.

Subglottic growths will, in all probability, call for an early laryngectomy.

Extent of Invasion.—So much depends upon the extent of the invasion that this element does in most cases dominate the plan of treatment. A small lesion in whatever part of the larynx of any age is likely to call forth a desire to treat it by very conservative methods, as the extreme methods which would eradicate the affection are so mutilating. It is always granted that the eye cannot tell the extent of deeper invasion, as a small pinhead-looking growth on the edge of the cord has frequently proven only to be the superficial evidence of an extensive invasion of the laryngeal wall or subglottic tissue. The function of the cord may point the way but does not do so in all cases, as many well developed growths may allow of free movement.

If after an attempted removal of the cord or part of the cord there is an early evidence of further growth, a laryngofissure leading to fulgeration or a hemilaryngectomy can be attempted but most surgeons are not wildly enthusiastic about the results from hemilaryngectomy.

The window resection of the cartilage of one side for the purpose of exposure to the X-ray or radium, it seems to me, to be ill advised, as an early case should be more radically operated upon,

and in late cases it opens the soft tissues to immediate invasion. Here a laryngectomy is in order.

When economically possible, all cases that show a lesion that has passed over the median line should be subjected to laryngectomy. There are inoperable cases, however, where it becomes necessary for the surgeon to choose some method of relief for the later distressing symptoms. Up to this time the tube has been resorted to nearly entirely, but I have called attention to the fact that by means of fulgeration we may be able to keep these patients much more happy for months or years at a time, the intralaryngeal application being given by means of a local anesthetic with very little discomfort. I have one patient who has been carried for one and a half years now by this method, and another for a year, and both are able to talk and live normally without the extreme inconvenience of the tube.

Social and Economic Condition of the Patient.—The practice of medicine is not based alone on the pathologic lesion. Hard and fast rules cannot be formed for guidance in treatment. The fact that a man has a carcinoma of the larynx cannot alone be used as an argument for a laryngectomy. The laryngectomized patients are many of them economically helpless. They may cough on least exertion and find that extended work is impossible. If they can do light work that requires more intelligence than hard labor they are lucky. But put such a patient out on the farm and he is nearly always lost.

The choice of treatment, therefore, for a financially crippled individual with no one to care for him but the State becomes a very serious problem. I believe in all these cases that a bold attempt should be made to handle these cases without a laryngectomy and without a tube if possible. Here fulgeration, X-ray, radium and minor operative procedures, intralaryngeal by preference, should be depended upon as much as possible. In all other cases, especially the intrinsic cases that do not come under classifications already mentioned, an early laryngectomy should be done.

Radium.—For many years radium was given up as a successful means of coping with this difficult affection, but of late there seems to be a tendency on the part of the radiologist to resume

treatment, a crossfire being used with massive doses from the outside repeated at intervals of two days so as to catch the growth with a series of attacks at a time when the maximum effect of the last application is being experienced. The local effect on the normal tissues is disregarded. If the radium is used intralaryngeally, a short exposure with the unscreened metal is indicated, this being succeeded by the crossfire in a few days.

490 POST STREET.

The Cause of Otosclerosis.

INTRODUCTION.

LOUIS K. GUGGENHEIM, M. D.,

ST. LOUIS.

In a preliminary report¹ of the present research, the writer stated that the footplate of the stapes, instead of being formed by the stapedia ring (derivative of Reichert's cartilage), is in reality derived from the labyrinthine capsule.

Priority in this observation was claimed because the textbooks to which the writer had access at the time described the footplate as a derivative of Reichert's cartilage. Since the preliminary report was made it has been ascertained, however, that Gradenigo² reported the true origin of the footplate in 1887. The significance in otosclerosis of the footplate returning, as it were, to the source whence it sprang will become apparent when the theory of regression is explained.

Suffice it to state at this time that through a study of the ears of certain lower vertebrates, having neither oval nor round windows, through the knowledge that, in otosclerosis, there occurs in typical cases a closure of the oval window (bony ankylosis of the footplate), and through a study of the pluripotential mesenchymal cells which represent the origin of capsular bone, the writer has come to believe and the purpose of the present research is to prove, that otosclerosis is directly the result of a regression in certain individuals through secondary mesenchymal activity, to a lower vertebrate level, as far as the aural capsule is concerned. After the aural capsule has completed its normal development, it will be shown that there remain in all individuals undifferentiated pluripotential mesenchyme cells in the region of the oval and round windows, canal region and neighborhood of

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the internal auditory meatus; that normally these cells rest, whereas in individuals whose chromosomes carry genes of regression there occurs a secondary mesenchymal activity leading to the condition called otosclerosis.

Investigators of the causes of otosclerosis have striven:

1. To reproduce by local experimentation the histologic picture of otosclerosis and to reach conclusions therefrom as to cause.
2. To deduce from a study of the microscopic and macroscopic otosclerotic ear the etiology of the condition.
3. To link otosclerosis with certain generalized bone pathologies.
4. To connect otosclerosis with endocrine abnormalities and to influence the otosclerotic patient by endocrine therapy.

Histopathogenesis of otosclerosis has been studied more extensively in Europe, for two reasons: Firstly, intimate knowledge of the histology and pathology of the temporal bone is almost hereditary among otolaryngologists in the great medical centers of Europe; secondly, the wealth of accumulated otosclerotic material in Vienna, Berlin, Hamburg and elsewhere in Europe offers the most splendid opportunities for study. American workers, on the whole, have been more interested in therapeutic experimentation. A remarkable admission has been made by every European worker in the field: After presenting his theory of the cause of otosclerosis, the investigator invariably adds, in effect, "But there is yet another factor, some constitutional, hereditary thing, which has not yet been revealed."

The program of the present work is as follows:

1. Presentation of the more important contributions with a critique of existing theories.
2. Explanation of the theory of regression (gene-propelled secondary pluripotential mesenchyme activity).
3. Phylogenesis of the ear.
4. Consideration of the development of the otic capsule from condensed mesenchyme to adult form (original microphotographic illustrations).
5. Histopathology of otosclerosis.

Inasmuch as the present work has for its purpose not only the presentation of the theory of regression but a comprehensive

analysis of the entire problem of otosclerosis, it must needs be published serially.

In reviewing the various theories of otosclerosis the writer will quote freely from the various publications, in most instances utilizing the author's own words.

Inasmuch as many of the articles have appeared in foreign languages, it is possible that the translation may be imperfect, and certain misrepresentations of the work of others result. Although great care has been exercised to avoid this, it would seem advisable for the reader to review the publications in the original form.

K. Wittmaack,³ in his article "Pathogenesis of Otosclerosis" states: "In the first place, the insufficiency of knowledge lies in the fact that the disease is peculiar and not identical with pathologic processes in other parts of the skeleton: I am therefore of the opinion that to compare it with other bony processes of the human skeleton is not of value in research into its pathogenesis."

"This peculiarity of otosclerosis depends on some special property of the bony structure of the labyrinthine capsule. The labyrinthine capsule attains its final structure and size early in life and normally does not undergo further transformation. Consequently, the distribution of the blood vessels, which is determined by the points of ossification, also remains practically unchanged; the different regions supplied by these blood vessels are more or less separated from one another, and new anastomoses cannot develop between them in later life. These peculiarities of the bony capsule are found not only in man but in other mammals, as, for example, in animals generally used for experimentation (guinea pig, rabbit), and even in certain nonmammals—i. e., birds. Thus by investigating pathologic processes in the bony labyrinth of these animals one may be able to expedite the interpretation of otosclerosis in man. In animals, however, spontaneous diseases of the labyrinthine capsule similar to otosclerosis have not yet been observed. Nevertheless, for many years I have found pathologic processes in the labyrinthine capsule of the hen which show a striking similarity to human otosclerosis."

He then reports the results of his experiments, the first being published in 1919. By injecting ferric chloride into the lumen of

the intracranial median sinus, circulation was stopped and sufficient venous stasis was usually produced to bring about the bony changes described. In 80 per cent of the experiments the positive result was evident, in 10 per cent it was somewhat uncertain and only in 10 per cent a result was not obtained. The author claims that all pictures of otosclerosis in the human being that have been published may be compared with the analogous pictures experimentally produced. He states that there is an anatomic difference between the bony capsule of man and that of the hen, particularly in regard to the distribution of blood vessels.

Many interesting photomicrographs showing otosclerosis in man and experimental changes in the hen are presented.

He ends with the thought that inasmuch as there is a striking similarity between the experimentally elicited condition and those found in human otosclerosis, we may conclude that the bony changes in man may be traced back to the same or, at least, a similar cause and that the pathologic process may be regarded as caused by the stasis or by the disturbance of the normal relation of hydrogen and hydroxyl ions within the tissue, closely contiguous to the vessels involved. Further: "No doubt, as a consequence of the stasis, the quantity of the H. ions increases within these regions of the bony tissue, and it appears that the bony substance responds by a typical biologic reaction. The manifestations of the process in the culminative stage may be interpreted as consequences of halisteresis."

The author then adds: "In spite of this knowledge, the problem of otosclerosis is not solved. The question now arises as to the cause of the venous stasis."

He believes that in otosclerosis there is often an hereditary factor but not always. He believes that the reactions of the vessels may result from an inherited inferior functional quality in the vascular system. He calls attention to the fact that, as a rule, the chief vein that leaves the oval window region is embedded in rigid walled surroundings and that this anatomic condition may cause a disturbance of circulation in the region involved.

In his article entitled "Die Ursache der Otosklerose," K. Wittmaack⁴ states that his belief in the venous stasis theory is based

upon the definite morphologic findings in actual cases of otosclerosis and upon the results of experiments in the hen. Every stage of histologic change demonstrable in actual otosclerosis this investigator claims to have produced upon the hen. He states that the hen experiments prove that the general venous stasis of the veins draining the capsule area causes the same biologic process that underlies otosclerosis. This indicates with great certainty, he adds, that the underlying cause of clinical otosclerosis must be a stasis within certain veins. He admits that the experiments throw no light upon the causative stasis in otosclerosis. Injections in monkeys have failed to clearly show the veins draining the capsule; therefore we must depend, he states, upon the further study of serial sections of human temporal bones. There follows the report of three cases of typical otosclerosis in the neighborhood of the oval window niche—two with a moderately marked involvement and one with a relatively small focus; in addition, a series of normal temporal bones. He found the veins draining the otosclerosis foci markedly distended by comparison with those of the normal specimens. From this he concludes a marked venous stasis exists in the otosclerotic foci. The author then gives a very detailed account of the veins which drain the oval window area and then, after having repeatedly stated venous stasis is without doubt the underlying cause of the capsule changes we call otosclerosis, makes the following statement: "Now in order for otosclerosis to really develop on the basis of venous stasis, there must be present, without doubt, still another factor. This is surely only the case in a certain per cent of all individuals, namely, in those who show the peculiarities of the venous channels." Then follows, "I am of the opinion that this factor is an exquisitely individual, mostly hereditary, predisposition which at this time we are unable with certainty to determine."

First there occurs a periodic stasis which gradually becomes permanent, after which a reversal of the current occurs. This throws the blood back into the bone at the site of predilection. He calls attention to the theory that varicosities of the legs are not due to mere stasis but to a reversal of the current in the veins. The varicosities in pregnancy he feels are due to the same thing plus an endocrine influence upon the vein walls, and further, that

the increased activity of otosclerosis during pregnancy is probably also due to the effect of internal secretions upon the ear capsule.

After comparing varicose veins of the legs to stasis in otosclerosis the author then states there may be no relation. The varicose veins, he adds, are due to some inherent abnormality of the vein walls; otosclerosis to unusually large communications between the predilection site veins with the carotid plexus or the middle meningeal artery plexus, or "There may exist in otosclerosis even another factor of which we have no knowledge whatever." He then adds the astonishing conclusion that although formerly he believed that a marked venous stasis was necessary to the production of otosclerosis, he now believes that even without this gross stasis and as a result of individual disposition otosclerosis may develop. He says we have learned through the years, through clinical observation, that otosclerosis is bound up with individual disposition—hereditary predisposition. Even though we see cases of otosclerosis which give no positive familial history, this cannot speak against the certainty of the hereditary factor, and it is often impossible to follow through generations a correct anamnesis.

He admits that the hen shows a much thinner lamellar covering of the enchondral parts and that the veins pass out in no such definite or fixed manner as out of mammalian capsules. Further, that immediately after leaving the capsule the veins of the hen pass into a large venous sinus which does not exist in mammals.

Wittmaack has been unable to produce changes in the monkey similar to those produced in the capsule of the hen. He has tried his experiment upon from sixty to seventy monkeys and all failed. He believes his failure has been due to the much greater thickness of the lamellar layer, which makes it impossible to produce stasis as deeply as the enchondral layer.

He feels he has gone as far as he can go. Further progress he feels can only be made on the living patient and that, he says, is impossible. He adds that possibly the extension of otosclerosis can be controlled by stopping the current of venous blood which is directed toward the capsule by excluding the veins involved. He suggests the possibility of opening up the squama, elevating the dura along the pyramid up to the emergence of the veins

involved. By this operation the veins will be torn by the elevation alone. Those that remain in the semicanal of the petrosus major may then be destroyed. The middle ear remains uninjured and unopened. The operation is described as follows: General anesthetic, the squama is exposed, a small trephine opening is made, dura exposed, separated and pushed upward. One sees the sulcus of the nervus petrosus superficialis major, sometimes minor; in the half-canal of the nervus petrosus superficialis major runs the vein. One finds also the vessels which run from the facial canal as well as such veins which come out of their own small openings in the bone. These veins are severed. If necessary one may elevate the dura still more, so that the middle meningeal artery is visible where it leaves the foramen spinosum. Behind it is found the sulcus for the nervus petrosus superficialis major, which runs toward the foramen lacerum: thrombosis of the veins follows.

Critique: One can only have the most profound respect for anything published by K. Wittmaack, not only because of his very interesting experiments upon the hen but because of his other numerous noteworthy contributions to otolaryngology. There is no doubt that Wittmaack has experimentally caused in the hen, through venous stasis, a resorption of capsular bone and that there has followed a filling in with mesenchyme, fibroblasts and then fiber-bone. Furthermore, the picture produced does suggest that seen in otosclerosis.

We must, however, keep in mind that the structure of the hen's capsule is different from the human; that the experimentally produced condition is always generalized as to the capsule, whereas otosclerosis occurs at certain sites of predilection; that except for the presence in otosclerosis of certain vessels gorged with blood corpuscles, there is no proof for the statement that venous stasis exists in this condition (in any very active bone change enlarged vessels are physiologic). Even admitting that the gorged vessels bespeak venous stasis in otosclerosis and that the experimental venous stasis produces a bone picture similar to otosclerosis, the writer feels that Wittmaack's experiments have thrown no light upon the basic cause of the human malady. Wittmaack frankly admits this when he states: "In spite of this knowledge, the problem of otosclerosis is not solved. The question now arises as to

the cause of the venous stasis." Although formerly he believed that a marked venous stasis was necessary to the production of otosclerosis he now believes that even without this gross stasis and as a result of individual disposition otosclerosis may develop.

Wittmaack has failed to produce any bone changes experimentally in monkeys.

We submit that it is common knowledge that early cases of otosclerosis show large vessels which contract in later stages; that these enlarged vessels may be physiologically due to increased bone activity, and that therefore Wittmaack has failed to prove that there exists venous stasis in otosclerosis; that the two types of capsules in hen and man are not comparable, and finally that Wittmaack himself has finally realized that, stasis or no stasis, there is an hereditary factor in otosclerosis which he has failed to uncover.

When he states that the bone changes in otosclerosis are peculiar to that malady and therefore nothing is to be gained by a study of bone processes elsewhere in the body, we must disagree. Of the osseous components of the otic capsule, namely, braided fiber-bone, lamellar haversian bone (osteon), lamellarless strand bone and ossified cartilage, the two first mentioned appear elsewhere in the body; further, in osteodystrophia fibrosa, a generalized bone pathology, we find resorption of lamellar bone and filling in with first, mesenchyme, then fibroblasts, and finally fiber-bone just as in otosclerosis. Therefore we must agree with Moritz Weber that much is to be gained by the study of such generalized bone pictures as in osteodystrophia fibrosa, rachitis, osteogenesis imperfecta, etc.

Inasmuch as we know that the histologic picture of osteodystrophia fibrosa and that of otosclerosis are practically identical, although no one claims that the underlying cause is the same, we submit that even had Wittmaack succeeded in producing in animals with otic capsules similar to man an otosclerotic picture by means of venous stasis (and he has not), we would still find it difficult to believe that the underlying cause would necessarily be identical in the two cases. Neither Wittmaack nor any other investigator has found in human otosclerosis any absolute evi-

dence of venous stasis. The enlarged vessels in otosclerotic foci are most probably due to the enormous bone activity in a normally resting area. In late stages these enlarged vessels grow smaller.

Wittmaack, basing a conclusion upon an unproved premise, states that the unknown hereditary factor in otosclerosis must be some inherited abnormality of the vessel wall. Until some more substantial proof of venous stasis in otosclerosis is forthcoming we feel that it is sufficient to state that there exists an unknown hereditary factor.

Comparing the varicosities of pregnancy to the theoretical stasis in otosclerosis we feel is far-fetched, to say the least.

Finally, Wittmaack's description of a craniotomy which has for its purpose the ceasing of a theoretical back-flow of venous blood into the capsule, is not only entirely uncalled for in the present state of our knowledge of otosclerosis, but represents a real menace, as here and there some uninitiated otologist, unable to appreciate Wittmaack's "anatomic phantasy," will most probably try out this operation upon some patient.

Finally, when Wittmaack states he has gone as far as he can go with his investigations, one is stimulated to analyze the "otosclerosis state of mind" which exists pretty generally throughout the world. The custom has been for each investigator to stick to his original theory through thick and thin. By prolific publication of his specific theory and by harsh criticism of every other theory, each investigator hopes to finally force the world of medicine to accept his ideas as the only true explanation of the condition. One outstanding exception to the above is Moritz Weber. Weber frankly admits he does not know what causes otosclerosis, but his broad scientific attack is what is needed to finally ascertain the true etiology of the condition.

Cl. F. Werner⁵ states that after the second year of life the bony capsule of the labyrinth neither grows nor changes its structure. As evidence of this he mentions the cartilage rests which remain to the extremest old age. Also, he concludes, the division of blood vessels shows no further change. Next he describes the process of ossification (anbau) and degenerative processes (Abbau) observed in the human labyrinth capsule.

He quotes the findings of Eckert-Möbius (1926) and M. Meyer (1931) that the endosteal layers of capsule bone show dehiscences and adds that these are not accidental variations but are the direct result of the laws of growth. The three canals show during the last fetal months at the periphery, resorption, and on the central wall, new-bone formation. There results an enlargement of the canal lumen and change in the arc form. As a result of the peripheral resorption, cartilage rests are found quite near the lumen; Howship's lacunae are common, giant cell osteoclasts are numerous, etc. In some areas (anterior vertical canal and posterior vertical canal), near the crus communis (posterior pyramid wall) the bone is only 20 to 30 mikron thick. Externally, under the dura, are found giant osteoclasts in large numbers. Resorption areas are sometimes only 10 mikron from the canal lumen. No cartilage rests are found in these thin areas. In three to four months fetuses the cartilage is here 200 to 300 mikron thick. As to the cochlea, the endosteal layer is absent at the apex. Here the cartilage rests encroach directly upon the lumen and are very numerous. Osteoclasts are rarely found on the inner capsule wall. The cartilage rests sometimes protrude into the lumen of the cochlea, deforming the membranous structure. The resorption of the inner wall would seem to result from pressure exerted by the membranous labyrinth. He quotes O. Mayer (1917) as stating that the endosteal layer is without a break and may be called general-lamella. Werner states that the endosteal layer is not continuous and not lamellar at all. Whereas, in man the labyrinth is covered by a compact bone-block which does not envisage the form of the labyrinth, in the hen the cranium is extensively pneumatized and the labyrinth is surrounded by a spongy network of trabeculae. Immediately surrounding the labyrinth is a compact bone in the form of the labyrinth. Ossification follows the formation of a cartilaginous model and begins in centers as in man. At the end of six months the bony capsule is complete. The conversion of cartilage into compact bone only takes place in the immediate vicinity of the labyrinth spaces; the remainder of the capsule is spongy (pneumatized). The spaces are lined with a connective-tissue bone. The canals for blood vessels are larger

and more numerous than in man. Cartilage rests are found at all ages (up to four years in this arbeit).

The same increase in the size of the canal lumen through resorption at periphery and increase of bone centrally was observed as in man.

Concerning the relation of otosclerosis to the experimentally produced hen picture, Werner asks:

1. How does the hen capsule develop and how does it compare to man?

2. Are the changes described by Wittmaack actually the result of stasis?

3. How does the stasis picture compare to otosclerosis? Is otosclerosis due to stasis?

Wittmaack used full grown hens where large marrow spaces are no longer present. The stasis produced the enlarged spaces. Hen and man develop capsules out of cartilage. Both show three layers. The enchondral layer shows small blood vessels and blue cement lines. Differences between hen and man: in the hen, the superficial layers of the cartilaginous capsule change into spongiosa, therefore the final actual labyrinth capsule is thinner than in man and its outer layers are supplied by the endost of the spongiosa spaces. There are about the cartilage rests more cement lines than in man. In man the original vessel regions remain unchanged; therefore, stasis would cause a very circumscribed bone change. About the oval window the periosteal capsule is very thick and compact, and the veins must pass quite a distance and isolated before emptying into larger venous spaces. Otosclerosis is not apt to occur where the firm lamellar bone has developed (periphery of canals), but in the enchondral regions where the original vein system remains unchanged.

Werner in his summary states:

1. The enchondral capsule suffers changes from degenerative (abbau) processes on the outer surfaces and near the lumen of the labyrinth.

2. In the last fetal months and first year the lumen of the canals is pushed outward by peripheral Abbau and central Anbau. On the periphery the enchondral capsule may disappear and be replaced by periosteal bone.

3. The endosteal layer is not closed but may be found open on such places as periphery of canals and cochlear apex where Abbau has occurred.

4. The resorption on the inner wall occurs without giant cells and is characterized by the great resistance of cartilage rests.

5. Growth processes result in only a superficial Abbau of the enchondral labyrinth but do not affect the inner enchondral layer. For the most part, this is equally true in the hen and man.

6. In the hen the enchondral ossification is finished at the ninth month. Cartilage rests remain throughout life.

7. The inner layer of the labyrinth capsule, consists as in man, of compact bone with narrow blood channels. This layer is, however, surrounded by spongy trabeculae.

8. The experimental "otosclerosis" of Wittmaack produced in adult hens cannot be confused with a stage of normal development but actually represents a pathologic process.

9. The normal histologic findings in man and hen speak not against but for a stasis theory. Otosclerosis in man is absent there where enchondral layers have disappeared through growth processes. That the pathologic changes in the hen are chiefly not focal but include the entire capsule is explained by the peculiar venous channels of the capsule.

Werner agrees with Wittmaack that the stasis picture in the hen is identical with that of otosclerosis, and that otosclerosis is due also to stasis.

Critique: Werner's article is most instructive because it so clearly describes the differences between the capsule of the hen and that of man, and rather substantiates the statement that pathologic processes in the two are hardly comparable.

Otto Mayer,⁶ in his article entitled "Die Entstehung der Spontanen frakturen der Labyrinth kapsel und ihre Bedeutung für die Otosklerose," first outlines the theory of G. Brühl as to the cause of otosclerosis. Brühl believes that a mechanical irritation of the area anterior to the oval window results from (1) constant movement of the annular ligament, and (2) the contractions of the tensor tympani muscle. Then follows Mayer's argument against this theory. Against: (1) "If it were true that the new bone formation is the result of the mechanical irritation from the annular

ligament, one would expect that the new bone formation would begin in the cartilage layer. I possess four temporal bones with beginning new bone formation on the anterior border of the oval window, in which the cartilage layer is perfectly normal, the new bone being anterior to it and impossible of irritation from the pull of the annular ligament." Proof against point (2): "The contraction of the tensor tympani muscle cannot irritate the point of predilection, as the muscle has no relation to this area. Just anterior to the oval window the muscle lies in a canal lined with thick, firm connective tissue in which the muscle and tendon move freely. The tendon passes around the processus cochleariformis, which should be the irritated point, and the processus cochleariformis shows no otosclerotic changes nor does the bone to which it is attached. Also, Brühl's theory could not explain otosclerosis in the region of the round window, internal auditory meatus and region of the canals."

Mayer studied thirty temporal bones of congenital deafmutism and found in these bones typical foci of otosclerosis; further, in otosclerosis the same changes to a lesser degree, were found in the inner ear as in congenital deafmutism. In examining the temporal bones of congenital deafmutes, Mayer found fissures which resembled those he had found in Paget's disease and which he termed spontaneous fractures.

He has found these spontaneous fractures in sixty series of temporal bones. These fissures are not empty but filled with fibers, poor in cells and partly ossified.

Mayer's theory of spontaneous fracture includes the entire pyramid, while Brühl's mechanical irritation theory concerns itself with the oval window region alone. He quotes the report of Looser, who in 1922 declared that undue strain on bone may produce, not a fracture, but a transformation which consisted in resorption of lamellar bone and the formation of a fibrous marrow in which a basket-weave bone tissue develops. This occurred in foci (Looser's Umbauzonen). Looser called this a kind of callus formation. He found in rickets and osteomalacia certain light areas in the X-ray which proved microscopically to be areas in which lamellar bone had been destroyed and fiber

bone formed in its place. Mayer believes exactly the same histologic process occurs in otosclerosis. Spontaneous fractures were found in old pyramids. The same mechanical strain occurring in the young could cause otosclerosis. The spontaneous fractures probably have no direct bearing upon otosclerosis. The relationship is that both occur in those areas in which the greatest strain occurs. A possible cause of physiologic injury Mayer sees in chewing.

Critique: There seems no longer any doubt that Mayer's spontaneous fractures are really fissures occurring during life and not artefacts as some have claimed. The presence of fibrous tissue in the fissures would seem to settle that question. That these fissures should occur in elderly people at the site of predilection for otosclerosis is certainly of significance. Many Americans are under the impression that Mayer has claimed that these spontaneous fractures are the cause of otosclerosis. Mayer has offered no such theory. He believes that in young bone, the strain, which in the old bone results in fracture, sets up sufficient disturbance, in predisposed individuals, to cause certain bone changes which ultimately result in the histologic picture of otosclerosis. This may well be true, but Mayer's observations, while scientifically of great interest, do not reveal the otosclerotic secret of the predisposed individual.

Mayer's observation of otosclerotic foci in a number of deaf-mute temporal bones is most interesting. Deafmutism of the congenital variety is the end-result of a chromosome transmitted abnormality or of an independent developmental anomaly. In such a temporal bone certain parts may be normal, while other parts show the widest divergence from the normal ear. In otosclerosis, the writer believes there is also a chromosome transmitted abnormality in the form of a certain cellular potentiality which will be discussed later.

Concerning Brühl's theory, which Mayer attacks, one might say that even though the annular ligament does not impinge directly upon the bone in which otosclerosis begins, it may well be that there is a constant though slight irritation transmitted through the cartilage, which irritation may be related to otosclerosis. Also in the case of the tensor tympani pull there may

be a transmitted irritation. Brühl claims only that these constant irritations may be sufficient to start otosclerotic bone changes in susceptible individuals. Neither Mayer nor Brühl has thrown any light upon that individual peculiarity which makes the development of otosclerosis possible.

A most accurate research into the finer structure of the labyrinthine capsule has been carried out by Max Meyer, so we must give careful consideration to his contributions.

Max Meyer:⁷ Specimens stained with hematoxylin and eosin, H. alone and E. alone, the fibers with anilin-water-gentian violet, silvered after Bielschowsky-Maresch, unstained specimens in ordinary and polarized light, were all placed side by side for study. With H. and E., bone spaces as well as vessels showed a homogeneous blue wall of about the thickness of a lamella. By high magnification one can often see that this wall is not homogeneous but made of small blue specks; probably blue stained very small calcium crystals. This is the same as in the cement lines. The red staining bone is strand bone; the gray-blue staining, true lamellar bone.

Blue coats are seen about very small vessels normally. The red staining bone is the embryologic fine fibered lamellarless bone (Max Meyer.) The differentiation between the lamellarless red staining strand bone and the lamellar gray-blue staining bone can best be made by staining one specimen with eosin, the next with hematoxylin. The calcified cartilage rests also stain blue.

Under oil immersion the blue stained bone shows between the lamellae lines of cement substance. In the gray-blue portions (lamellar bone and cement substance) one sees, under oil immersion, blue granules (calcium granules). The granule portion stains deep blue and is the essential component of the lamellar bone for blue color reaction. There is no room in the lamellarless strand bone for cement substance.

In unstained specimens with polarized light the lamellarless bone shows as bright silver, while the lamellar system is poorer in fibers and shows as black and white.

The periosteal layer of the capsule shows three differently stained parts with H. E.: (1) blue to gray-blue coats around certain vessels; (2) a red area around the coats and also around

the small vessels; (3) an irregularly constructed bone tissue of violet to gray, also often red.

The coats (1) consist of typical lamellar bone. They correspond to those (much fewer) in the enchondral portion. In accordance with the slight metamorphosis in this portion of the capsule, these blue bone areas are found also frequently in the resorption spaces and in postembryonal closure of marrow spaces. The blue resorption space linings show cementum. The blue linings date back to the period when osteoblasts laid down lamellar bone, namely, after the first year of life.

To Max Meyer, it appears that in early life the lamellar system seems oftener to show a richer fiber formation and a poorer cement content, while in older specimens the condition is the reverse; therefore, in early life the specimens stain less blue than later.

At the end of the embryonal period the perichondral portion of the capsule is built of a network of braided (*geflechtartigen*) bone with open marrow spaces of varying size. If the meshes are very closely woven, then this tissue bounds the vessel canals throughout life. We may also see next to the vessels a more or less thick layer of fine-fiber bone with parallel fiber arrangement (lamellarless bone, Max Meyer). It seems that the fibers of the two types of bone intermingle. Sometimes vessels are surrounded entirely by lamellarless bone. Sometimes in the second year, when osteoblasts are building lamellar bone, there remains between vessel and lamellar bone an area of lamellarless bone.

In H. E. specimens the lamellar coat of a vessel stains blue-gray, the lamellarless marrow bone, red to violet, and the braided bone a dark violet to gray (seldom red). Of the three types of bone the richest in fibers is the lamellarless fine-fibered (strand) bone, which is the special labyrinth bone of the adult (in embryos, such bone is temporarily found elsewhere). It stains most markedly red and remains mostly unstained in H. specimens. The fewer fibers, the bluer the bone will stain.

In the lamellarless marrow bone of the periosteal capsule the oxyphile element predominates, depending upon richness in fibers which leave less space for cement substance. The cement substance between lamellæ is basophil and stains blue with H.

The belief of Brunner that the decided blue staining of the bone in otosclerosis is due to an especial richness in cement substance wins in probability through the fact that for the normal labyrinth capsule, experimenting with various stains, the following has been noted: The lamellarless, fine-fibered strand labyrinth bone, which makes up the greater part of the enchondral capsule and is present in smaller amounts in the periosteal capsule holds many fibrils and therefore less cement substance and stains with H. E. after complete decalcification strongly red. All lamellar bone in the enchondral capsule surrounding small vessels and in the periosteal capsule holds more or less broad cement substance layers between the lamellæ and stains blue to blue-gray. The braided bone shows locally and individually varying amounts of fibrils and cement substance, mostly more of the cement substance. The fibrils in braided-bone vary in length and thickness. The fibrils of strand and lamellar bone are very slender and uniform. The particular character of the braided-bone fibrils probably plays a rôle in the staining, one time toward the oxy- and the other toward the basophil side. The braided-bone stains more strongly with H., as a rule, and shows as blue to violet to gray (colorless). Therefore, one may conclude that in deep-blue bone tissue in disease conditions we are dealing with more calcified cement substance, regardless of the kind of bone involved. Deep-blue bone can therefore theoretically be braided, lamellar or lamellarless fine-fibered.

We know that the labyrinth bone is the hardest in the body; further, that the hardness is due to the amount of calx contained. A large part of the capsule consists of a special bone tissue, the lamellarless, fine-fibered, strand-like marrow bone, particularly rich in fibrils and therefore with relatively small space for calx-holding cement masses. Max Meyer strives to explain this paradoxical situation as follows: through the compactness of the entire capsule bone so much calx is present that the bone is extremely hard, despite the fact that in certain parts less calcium containing substance is present.

THE CAPSULE IN RACHITIS.

Case 1.—Age 6 months; rachitis. Died of leptomeningitis purulenta. Both temporal bones showed development in accord with

age. The enchondral capsule showed a fairly compact structure. Cartilage rests as usual. Bone tissue, under polarized light and with fibril staining, showed over all a lamellarless fine-fibered, strand-like marrow bone. Enchondral ossification was almost ended. The cartilage mass in front of the oval window was completely ossified, right and left. In the right temporal bone no further process of ossification was observed. In the left temporal bone some areas of cartilage were undergoing ossification.

The periosteal capsule, right and left, anteriorly was very compact, while posteriorly it consisted of a large-meshed spongiosa with marked pathologic changes. In the compact portions was noted a network of gross braided-bone, in the meshes of which were vessels surrounded by deep blue zones. Occasionally the vessels were surrounded by a coat of strand bone. Lamellar systems were not found with certainty. In the porus internus, apex of petrous portion and in the entire mastoid region the periosteal bone picture was quite different. Under low power one saw in various areas of marrow tissue a narrow network of rose-stained osteoid with only a few blue-stained areas of calcification. The osteoid trabeculae showed in some areas braided bone structure; also strand bone was seen. Osteoblasts were present. The marrow spaces of the mastoid were fibrous in nature. About the antrum and between petrous portion and mastoid was lymph marrow. In the tympanum was evidence of O. M. P. C. undergoing organization. The ossicles showed a spongiosa picture with much osteoid.

Case 2.—Age 9 months; rachitis and bronchopneumonia. In the periosteal capsule on all spongiosa trabeculae was found uncalcified osteoid tissue showing strand formation and some braided structure. In the enchondral portion typical rachitic changes were found only in one small cartilage area. Pneumatization of the mastoid was inhibited through rachitic bone formation. The tympanum showed inflammation.

SUMMARY.

The skeletal changes in rachitis must be looked upon as a manifestation of a general disturbance of metabolism. The bony capsule shows, to the end of life, embryonal tissue. The chief

changes are in the periosteal bone. A disturbance of cartilage with ingrowing fibrous tissue has been observed. In the healing of the rachitis this fibrous tissue may become ossified.

Meyer believes that mechanical factors in rachitis, such as the pull on the mastoid by the S. M. muscle, may cause the change of lymph-marrow into fibrous. The large amount of osteoid represents an effort to make up in quantity what is lacking in bone quality. The inhibition of pneumatization was due to the otitis media, according to Wittmaack, but Max Meyer sees in this lack of pneumatization a result of the rachitic bone change, causing the spongiosa to become more and more compact.

OSTEOMALACIA AND SENILE OSTEOPOROSIS.

Case 1. Osteomalacia.—Age 71. Tympanum normal; marked pneumatization. In the trabeculae of the mastoid were resorption areas about vessels; these areas were filled with blood. Numerous marrow spaces in the cortex contained fat and showed hyperemia. The resorption areas showed lacunae but no osteoclasts. Osteoid trabeculae with small osteoblasts were found. Concerning the marrow spaces in relation to bone substance: Normally, at 71, the periosteal bone shows a certain degree of old-age porosity. In this case the porosity was very extensive, especially in the periosteal portion, but also in the enchondral parts. The marrow spaces showed typical fat marrow, with here and there remains of lymph marrow. No fibrous marrow was found. Everywhere the fat marrow showed hyperemia. The bone trabeculae were with H. and E. stained bright red with intermixtures of bluish bone. Pale rose trabeculae in apposition to osteoblast chains were noted. In polarized-light the new bone was lamellar; the old, lamellarless. Also lamellarless plus braided-bone, dating from the embryonic period, were seen.

Everywhere were single vessels surrounded by bright red osteons. In the osteoid trabeculae were occasional granules of blue calcium. The endosteal "general lamella" showed some degeneration and new bone formation (lamellar). In the region of the horizontal canal was an area where the endosteal layer was entirely absent with numerous lacunae and osteoclasts. Near by were osteoblasts and osteoid. In the posterior canal an osteophyte

protruded into the lumen. Many fractures radiating from the labyrinth cavities were seen. Most of these were artefacts, but some surely occurred during life. In two fissures, (1) between the round window and posterior ampulla, and (2) from vestibulum to a marrow space, a cement lining and loose connective tissue were found. No new bone was found in these fractures.

Case 2.—Age 72; senile porosis. Marked porosis of the capsule extending to the labyrinth cavities. Thickening of bone of the promontory with decrease in size of round window was seen. Marrow spaces showed fat, seldom fibrous tissue.

Nager and Meyer:⁸ Osteogenesis imperfecta congenita of the human labyrinth capsule.

Case report: Normally born child, died from intestinal hemorrhage on seventh day. Typical signs of O. I., namely, markedly shortened limbs; thickened, smooth, flexible skin; thighs contracted toward body; legs hanging limp; cranial dome absent; only part of frontal, temporal and squama occipitalis to be felt; expression of face old; sclera markedly blue; no struma; cartilage-bone appositions of ribs showing rosary formation; abdomen swollen and soft; edema of feet and labia majora; urine, albumin +; squamous epithelium; leucocytes.

X-ray: In all bones an increase in ray transmission; thinned cortex; spongiosa irregular; epiphysis lines straight and sharp, with plainly seen calcification areas. Numerous fractures with callus formation.

Skull: Base and facial bones gave a relatively good shadow. Cranial vault consisted of irregular small and large flecks and streaks of lessened density.

Right temporal bone. Labyrinth soft parts quite normal. Middle ear normal.

The ossicles, especially the malleus, showed definite scarcity of bone formation but were otherwise normal.

The bony capsule: First one noted an unusual compactness of the bone next the cavities. The more peripheral parts were very poor in bone, including both enchondral and perichondral divisions. The poorest area in bone was posterior to the fossa subarcuata. Under higher magnification the following abnormalities were seen:

Perichondral and endosteal bone fairly well developed. In the region of the canals, however, underdevelopment of bone was noted. In other places the marrow spaces were very small. Mostly braided bone was seen; only here and there lamellarless, fine-fibred bone tissue was present.

The enchondral capsule: An occasional massing of bone cells in the globuli ossei (bone extending into cartilage rests). Cartilage rests looked normal except at the apex of the cochlea, where they were more numerous and larger than normal. A large cartilage mass was found here, enclosed within the bone. The ground substance was not calcified. The endosteal bone was absent at the apex. Posterior to the posterior canal a large mass of cartilage undergoing change was found. Many of the pale cartilage cells showed no change. Scarcity of bone deposit was noted. No osteoid was seen. No fractures were found in the capsule.

(This is the first of a series of articles on otosclerosis by Dr. Guggenheim. The next will appear in an early issue.)

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Neoplasms of the Nose, Throat and Ear.

CARCINOMA OF THE TONGUE WITH SPECIAL REFERENCE TO TREATMENT BY IRRADIATION.*

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Carcinoma of the tongue is a sufficiently common occurrence to warrant serious consideration by the practicing laryngologist. Indeed it is to him that the general practitioner so frequently turns for aid when confronted by such a grave condition.

To those who are at all familiar with the literature, just another contribution on malignant disease of the tongue would add nothing to what has been established relative to our knowledge concerning this dire affliction. However, there have been many advances, especially within the realm of histopathology and irradiation therapy, that justify this contribution in order that this knowledge may have a more general distribution. In fact, these advances have had a profound influence upon our therapeutic approach in malignant disease of the tongue.

That all malignant tumors of the tongue do not pursue a similar course is a matter of common experience. Just why one lingual neoplasm will grow rapidly and cause the death of the patient within a period of a few months and another grossly similar mass progress but slowly and only cause death after an interval of a number of years, has caused a great deal of speculation in both the pathologist as well as the clinician. It is only since the work of Broders and McCarty that the study of epithelial neoplasms in attempting to relate their histologic picture to the clinical course that the disease pursues, has been popularized.

*Presented before the Section on Eye, Ear, Nose and Throat of the Illinois State Medical Society, at Springfield, Ill., May 17, 1932.

The classification in four groups as proposed by them has not received universal acceptance. As is well known, practically every histologic type of epidermoid carcinoma may be found affecting the tongue from the adult fully differentiated squamous growth to the more or less totally anaplastic transitional cell or lympho-epithelial variety. For practical purposes a division of lingual carcinomata into three groups has been acceptable, especially to the clinician. In the first group are found those neoplasms of comparatively slow growth that exhibit but little tendency towards metastasis to the cervical lymph nodes and the parenchymatous organs. Histologically these neoplasms show a picture of a more or less fully differentiated adult type of a neoplasm, the cell exhibiting elongated pavement like structure with relatively rare mitotic figures and a pronounced tendency towards the exhibition of keratinization, pearl formation and intercellular bridges. At the other extreme, and included in the so-called group three, are those neoplasms that exhibit a high degree of malignancy. In spite of their frequent small size they have a rapid rate of growth with a marked tendency toward early, wide, and diffuse metastasis and causing death within a relatively short period of time. Microscopically these lesions evidence a picture of an embryonal, anaplastic, undifferentiated growth. The neoplasm is highly cellular with the individual cells exhibiting marked variation in size, shape and staining qualities. Mitotic figures are abundant and adult features, such as keratinization, pearl formation and intercellular bridges are conspicuous by their absence.

Between these two extremes is found an intermediate group, the so-called group two. These have features that lie between the adult characteristics of group one and the anaplastic, embryonal features of group three.

Clinical experience has enabled us to draw several important conclusions regarding those neoplasms that may be found either in group one or three. Thus, it has been found that trauma has but a minimal effect in accelerating growth or producing metastasis, in the lesions that fall into group one, and, therefore, surgical intervention in the early localized stages of the disease, providing the anatomic location is favorable, is attended by excellent results. The effect of irradiation is comparatively mild and, there-

fore, these growths are relatively radioresistant. On this account irradiation therapy is frequently disappointing in these types of lingual malignancies. In the neoplasms in group three any trauma, no matter how insignificant, may accelerate growth or metastasis. Not infrequently local glandular, as well as visceral metastasis, may have occurred before the growth has reached sufficient size to be attended by clinical symptoms sufficient in intensity to cause the patient to seek medical advice. Surgery has been found to be attended by but poor results and is therefore contraindicated in these types of neoplasms. In many of these growths relatively small amounts of irradiation can produce dramatically rapid disappearance of the growth, and these tumors are therefore radio-sensitive. It follows that in consequence of the poor results with surgery and the spectacular response to irradiation that these neoplasms are best treated by radium or deep X-ray therapy. No such definite statements can be made relative to the neoplasms that are placed in the category of group two. Some may show characteristics that are predominantly of group one and are to be treated along the lines suggested for those neoplasms, and others may, of course, show characteristics of group three and should be treated accordingly.

In group three there have been isolated two types of neoplasms that are accompanied by a highly characteristic, unique histologic picture and are associated with extreme radio-sensitivity—the so-called transitional cell carcinoma and lympho-epithelioma.

The transitional cell carcinoma has a histologic structure of strands or sheets of round or polygonal cells with a markedly hyperchromatic nucleus. The lympho-epithelioma, histologically, is composed of a large number of relatively large cells, containing a very small pale staining rim of cytoplasm. The cell outlines are frequently difficult to distinguish and may give one the impression of a syncytial mass. The nuclei are large, occupying practically the entire cell and exhibiting a prominent nucleolus. Throughout the section one finds areas of infiltrations with lymphocytes that may at times be so dense as to practically obscure the underlying cell structures.

These neoplasms have all the characteristics of highly anaplastic malignant growths and are exceptionally radio-sensitive.

These histologic studies of the relative degree of cellular differentiation or of the potential malignancies of the growth have been most enlightening, from a practical point of view, in explaining the success or lack of success with surgery or irradiation in the various types of neoplasms so treated. These histologic criteria are important, not only in determining whether the tumor is best treated by surgery or irradiation, but also is a factor in calculating the intensity of the irradiation dosage. It is to be seen, therefore, that the treatment of lingual carcinomata cannot be placed in a routine standard clinical procedure, but becomes a special problem in each individual case. We have elaborated a dictum that the biopsy determines the details of treatment. These studies have also shown that the lack of success with surgery in attempting to treat the carcinomata at the base of the tongue was due more often to the fact that we were dealing with a highly undifferentiated anaplastic growth, rather than the technical surgical difficulties encountered, and conversely, the highly spectacular and dramatic regressions of apparently hopeless growths in this same region following irradiation are to be explained in the same manner.

Recent advances in surgical technic in the treatment of malignancies of the tongue are not many. The use of surgical endothermy has replaced the cold knife. However, we employ it only in small more or less localized lesions of the anterior portion of the tongue, that histologically fall into group one. As a precautionary measure, the lingual arteries are ligated previous to the attack with the electrothermic methods, as not infrequently an alarming secondary hemorrhage may occur.

Irradiation is one of the most important weapons at our command in the treatment of malignancy of the tongue. However, it must be employed, when indicated, in adequate doses, together with proper screening, as well as other technical factors that time will not permit of discussion. Irradiation delivered by direct surface application of only small amounts of radium is mentioned but to be condemned. Interstitial irradiation is highly successful. The best results are obtained by using small amounts of radium or radon, using a screen of at least .5 mm. of platinum or its equivalent over a long period of time. If radon is employed we

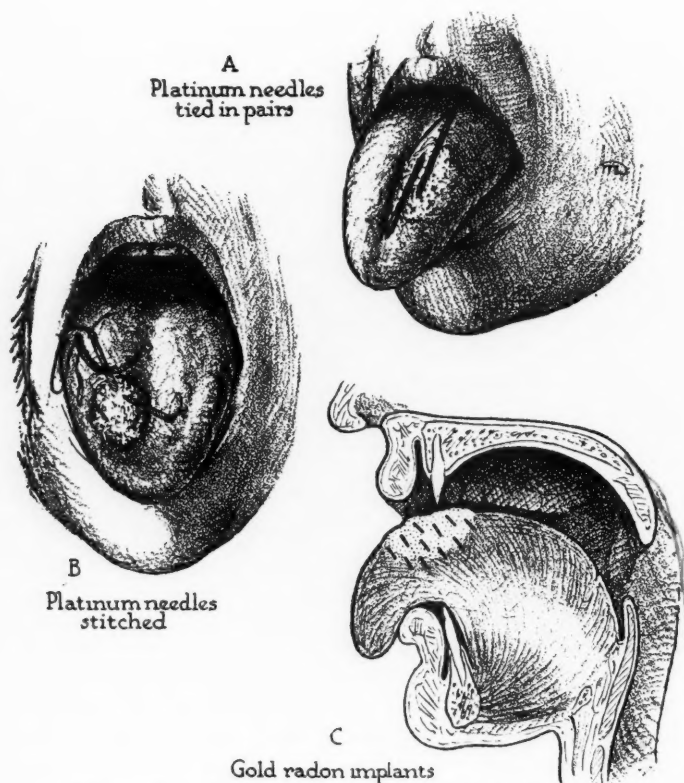


Fig. 1. Use of radium needle and radon seeds in the tongue.

advocate the use of the removable platinum radon containers in order that some of the disagreeable sequelæ on leaving the seeds permanently in the tissues may be obviated.

The method of implanting the needles or radon implants vary with the size, location and depth of the lesion. (Fig. 1.) Local anesthesia is effected by conductive injection of the lingual nerve, and in addition the mucosa over the area may be brushed with a solution of cocain. In small lesions the implants are placed in palisade formation about the circumference of the lesion at a small distance from its periphery without entering the tumor proper.

Larger lesions require the insertion of the needles into the actual tumor itself in addition to the surrounding palisade. Growths in the posterior portion of the tongue may be irradiated by inserting the needles or radon implants into it via the anterior inferior aspect of the tongue or through the skin just above the hyoid bone. A glove finger placed into the mouth on the back of the tongue acts as a guide to the proper placement of the implant.

One of the most important advances in the treatment of carcinoma of the tongue by irradiation is the use of large amounts of radium at a distance, or the so-called telioradium therapy. It consists in the use of one to four grams of radium at a distance of six, ten or fifteen cm. This technical advance bids fair to produce brilliant results, especially in the more radiosensitive types of growths. Unfortunately, the tremendous cost of such a large amount of radium, in the neighborhood of \$250,000.00, will prevent its use except in the larger clinical centers that have adequate financial endowment. We are indeed fortunate to have at our disposal at the Michael Reese Hospital a four gram pack or bomb (Fig. 2), which permits us to utilize this most efficient type of irradiation therapy. Lacking such technical means, the use of highly filtered deep X-ray therapy may be a more or less acceptable substitute, as it is far better to employ a good dose of X-ray therapy than a poor dose of radium.

The treatment of the neck, in association with carcinoma of the tongue is just as important as the treatment of the primary growth itself. It must be recognized that microscopic metastasis may be present in the glands of the neck without any palpable evidence. It must also be understood that not all palpable glandular enlargements are necessarily malignant, as a fair percentage are infectious. The therapy advocated for the neck is dependent upon the nature of the primary growth, the presence or absence of palpable metastasis, as well as their extent. If no glandular involvement can be made out grossly the prognosis is, of course, materially improved, especially if the primary growth is one of the adult fully differentiated types of carcinoma. In these cases the neck is subjected to intensive irradiation in any event, preferably by the use of a four gram pack at a distance. If a radium pack or bomb is not available, one may utilize highly filtered deep

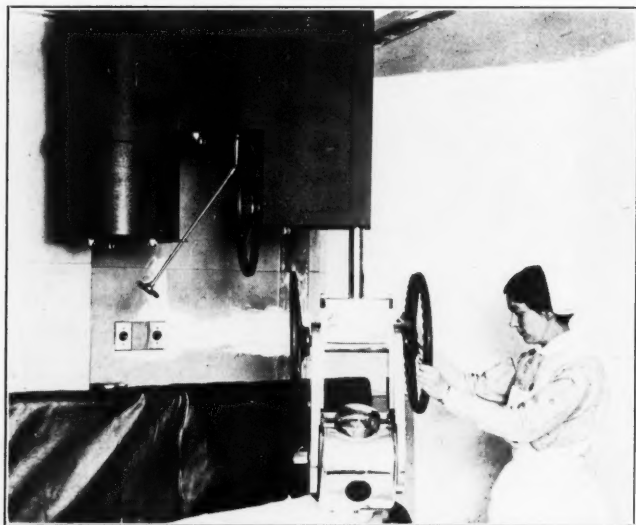


Fig. 2. Four gram radium bomb at the Michael Reese Hospital.

X-ray or one may systematically needle the neck by the subcutaneous placement of radium throughout the cervical area. Needles containing 1.3 or 2.0 mg. of radium, with a .5 mm. platinum screen are employed. After proper preparation of the skin and under gas anesthesia, the needles may be inserted along the anterior and posterior borders of the sternomastoid, from the clavicle to the base of the skull, below the mandible throughout the submaxillary triangle and just above the hyoid bone. This procedure is carried out on both sides of the neck at the same time. Care must be taken in the proper technical placement of the needles so as not to endanger the mandible, the hyoid bone, the thyroid cartilage and the important blood vessels of the neck. The needles are permitted to remain in situ for a period of from five to seven days. The resultant radiodermatitis, so well described by Regaud, is an index of efficient irradiation. This method of subcutaneous interstitial irradiation is preferable to the Columbia paste collars that are also advocated.

The presence of palpable nodes modifies the treatment indicated. While conceding that a good percentage of the palpable nodes may not be malignant in practice, all glandular enlargements are treated as if they are proven carcinomas.

If the primary lesion in the tongue is an adult type of tumor, a radical resection of the gland bearing tissues is performed. It is frequently necessary to resect the jugular vein with the involved chain of gland, and we have, on several occasions, even resected the common carotid artery on the affected side. Obviously but one internal jugular vein may be removed. A few days following the surgery upon the neck irradiation therapy, preferably with the four gram bomb is instituted. Lacking the necessary amount of radium, one may insert the needles as described above at the time of the neck operation, or if deemed advisable, at a later time. However, if the primary growth is of the anaplastic, transitional cell or lympho-epithelioma type, no surgery is performed and irradiation, as described above, is depended upon.

CONCLUSIONS.

1. The problem of therapy of carcinoma of the tongue has undergone radical changes within the past seven years.
2. Malignant epithelial growths of the tongue vary in their histologic structure, biologic course and response to irradiation.
3. Adult, fully differentiated carcinomata grow slowly, metastasis late, and are relatively radioresistant. They are, therefore, better treated by endothermic excision, if small, and localized to the anterior portion of the tongue.
4. Anaplastic, embryonic undifferentiated growths are highly malignant, metastasis early, and wide, and are highly radiosensitive, especially the transitional cell and lympho-epithelial variety. They are best treated by irradiation, preferably with the four gram bomb, and in association with interstitial radium or radon, if necessary.
5. The gland bearing area of the neck must receive appropriate attention in every case.
6. Where no palpable glands are present in the neck, prophylactic irradiation is necessary, preferably with a four gram pack.

7. Palpable glands, secondary to a fully differentiated primary growth in the tongue, are best treated by a radical gland resection in conjunction with irradiation.

8. Palpable glands, secondary to an anaplastic, undifferentiated primary growth, are treated by irradiation along and surgery upon the neck is contraindicated.

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DISCUSSION.

DR. M. REESE GUTTMAN, Chicago (closing): Unfortunately my associate, Dr. Beck, is not able to be present. He had fully intended in the discussion to bring out the fact that not one single case of a large series of malignancies of the posterior portion of the tongue that he had seen in thirty years of practice had remained well, prior to 1926. Since that time there have been several technical advances in the use of radium that has permitted a more hopeful outlook. A review of the literature emanating from the large radium centers throughout the world will show that something between 30 and 40 per cent of the growths in this region may be cured or at least held in abeyance for a period of over five years, and we have had similar good fortune in a small number of cases. Surgery, unfortunately, is followed by poor results, and, therefore, irradiation is the treatment of choice of cancer of the posterior third of the tongue. Surgical resection is only indicated when the growth is small and limited to the anterior portion of the tongue. The best results are to be obtained in the use of telioradium therapy or the so-called bomb. There have been recent advances in deep X-ray therapy with the development of machines that are capable of producing one million or more volts. In Germany, deep X-ray therapy machines of three and one-half million volts are said to have been engineered. The ray emitted from an X-ray tube subjected to three and one-half million volts closely approaches the gamma ray produced by the disintegration of radium. It is possible that in the future this type of deep X-ray therapy, which is purely experimental at the present, may be an important factor in the treatment of carcinoma.

The Scientific Papers of the American Bronchoscopic Society.

LXXII.

PAPILLOMA OF THE BRONCHUS.

HENRY BOYLAN ORTON, M. D.,

NEWARK, N. J.

The literature available on papillomata of the bronchus is very meager.

Ellen J. Patterson¹ had a very interesting article on multiple papillomata of the esophagus, published in the Transactions of this Society, 1927, with a complete bibliography, and mentions Chevalier Jackson reporting a case of single pedunculated papilloma of the esophagus.

John D. Kernan² has reported a case of papilloma of the lower end of the esophagus in a child, age ten years.

Simon Jesberg³ reported a case of a six-year-old boy with a diagnosis of right bronchial obstruction. On bronchoscopic examination a pedunculated tumor was removed, and the pathologic examination showed it to be a polyp.

Seigert⁴ reported autopsy findings of a papilloma at the bifurcation of the trachea, apparently filling it and projecting into the right bronchus.

Spiess⁵ found a polyp taking its origin from the right bronchus just below the bifurcation. The right bronchus was completely filled and the left partially so. The tumor was successfully removed by bronchoscopy.

Mervin C. Myerson⁶ reported two cases of polyp removed bronchoscopically and four observed at autopsy; also in a personal communication mentions a patient, a man, who had papilloma of the bronchus as well as papilloma of the larynx.

The report of the following two cases really brings out the value of diagnostic bronchoscopy in obscure respiratory dyspnea more than the finding, possibly, of the papilloma.

First case: Martin O'H., age 6 years, was first seen October 11, 1922, with marked laryngeal dyspnea. A diagnosis of papilloma of the larynx was made and a tracheotomy performed the same day. Subsequently a number of laryngoscopies were done with removal of papillomata.

One year later, October, 1923, the patient returned to the hospital because of respiratory dyspnea. Laryngoscopic examination did not reveal any obstruction in the larynx; then a bronchoscope was passed, and in the trachea, at the bifurcation, a papilloma was removed.

The patient improved until August 13, 1924, when he was readmitted to the hospital because of respiratory dyspnea, at which time a bronchoscopy was done with removal of papilloma from the trachea.

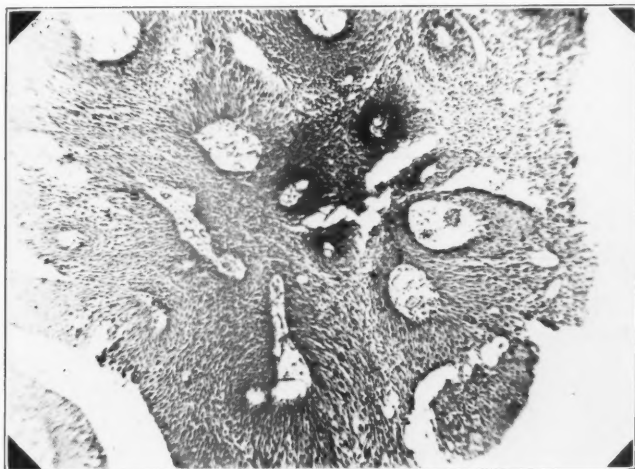
Subsequent bronchoscopies extending over a period of three years did not reveal any additional papilloma.

Second case: Melvin R., age 3 years, was referred to the hospital May 22, 1929, because of obstructive dyspnea and a diagnosis of papilloma of the larynx was made. A tracheotomy was done the same day by Dr. Wood.

Past history: Child contracted a cold at the age of six months; developed whooping cough at the age of fifteen months. The mother stated the child's voice had not been normal since that time; also had difficulty in breathing, which had become worse two weeks prior to entering the hospital.

Numerous laryngoscopies were done with the removal of papillomata, and the patient was decannulated nine months later.

The patient remained well until March, 1931, when he developed measles with an annoying cough. On November 18, 1931, one year and nine months following decannulization, the patient was readmitted to the hospital with the following symptoms: Cough, severe dyspnea, attacks of suffocation, temperature 104, pulse 116, respiration 36. Examination revealed: Larynx was negative; urine negative; blood count showed 70 per cent hemo-



globin; R. B. C., 4,300,000; W. B. C., 52,600, with the polys 92 per cent. The medical service was asked to examine the chest of the patient with a possibility of it being pneumonia. Their report was negative. X-ray of the chest did not reveal any opaque or nonopaque body, and on November 25, 1931, a bronchoscopy was done and a large papilloma removed from the left bronchus at the bifurcation of the trachea.

A subsequent bronchoscopy five months later, April 22, 1932, revealed a small papilloma in the trachea which was removed.

I am of the opinion that both of these patients were unquestionably transplants and not primary papilloma, but it does bring out very beautifully to my mind the value of diagnostic bronchoscopy as an aid in obscure chest conditions.

24 COMMERCE ST.

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LXXIII.

REPORT OF TWO BRONCHOSCOPIC CASES.

W. LIKELY SIMPSON, M. D.,

MEMPHIS.

1. X-ray Showing Watermelon Seed in the Trachea.—Master S. L. S., age 4, with a history of a watermelon seed having been inhaled two days previously with the usual symptoms, came to see me with an X-ray, showing the watermelon seed in the anteroposterior position in the trachea. There is nothing unusual about the removal of the foreign body, but I am presenting a slide of the roentgenogram of the watermelon seed in the trachea.



Fig. 1. Watermelon seed anteroposteriorly in trachea.

2. Removal of Nail Which Had Been in a Peripheral Bronchus for Four Years. — Jesse C., age 18, gave a history of having inhaled a nail four years previously. The patient had pneumonia twice and also had an occasional hemorrhage. Thirteen attempts at removal were made before the foreign body was seen and removed from the small bronchus of the lower lobe of the right lung. The foreign body was not seen at any attempt at removal except the last one. It was finally located in a small peripheral bronchus to the outer side of the lower lobe of the right lung. Only a 4 mm. tube could be used. The use of the fluoroscope was very helpful in the procedures.



Fig. 2. Bronchoscope and probe in bronchus near foreign body.

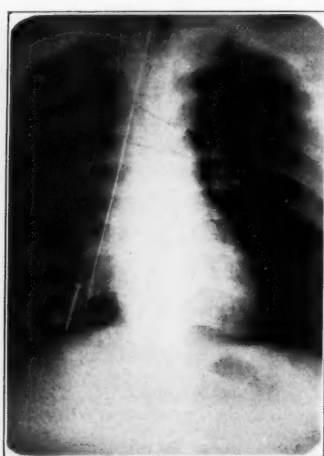


Fig. 3. Probe in bronchus quite near foreign body.



Fig. 4. Bronchoscope and probe in bronchus near foreign body.

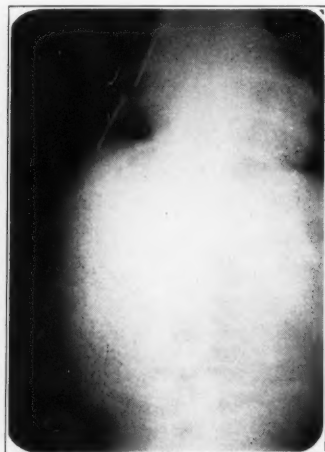


Fig. 5. Probe almost on foreign body.

LXXIV.

REPORT OF A CASE OF PARALYSIS OF THE LEFT SIDE OF THE LARYNX FROM A HYDROPNEUMO- THORAX OF THE RIGHT SIDE.

W. LIKELY SIMPSON, M. D.,

MEMPHIS.

Unilateral paralysis of the larynx is not a very uncommon condition and is usually due to pressure upon the left recurrent laryngeal nerve by aortic aneurysm, mediastinal growths, heart lesions, enlarged pulmonary artery, goiter, etc.

Herbert Tilley, in a paper, "Mechanical Immobilization of the Vocal Cords," speaks of the possibility of an immobile cord, being due to fixation of the corresponding crico-arytenoid joint, and in this condition usually the cord is only temporarily immobilized, having as its etiology gout, rheumatism, syphilis, etc.

St. Clair Thomson, in discussing Tilley's paper, said that he was inclined to the conclusion that articular causes of impaired movements were rare, and that neurologic causes were much the most frequent. In this same meeting Lambert, in discussing Tilley's paper, gave an analysis of 189 cases of paralysis of the vocal cords, compiled from the ear and throat department of the Royal Infirmary of Edinburgh. The patients were under the care of Drs. Turner and Fraser, 1907-26. One hundred eighty-one were unilateral and eight bilateral, forty-six right sided and 135 left sided. One of these cases had a left sided pleural effusion.

Garland and White, in a paper on "Paralysis of the Left Recurrent Laryngeal Nerve, Associated with Stenosis," said in discussing etiology, that the enlarged left auricle causes pressure on the left recurrent nerve, producing either a neuritis or a nerve atrophy. In their series of nine cases, case No. 4 was a female, aged 40, with a diagnosis of a right pyopneumothorax, mitral stenosis and left recurrent laryngeal nerve paralysis. The X-ray showed a hydropneumothorax on the right with a displacement of the heart to the left.



Fig. 3. Later stage than Fig. 2, after lung is filling.

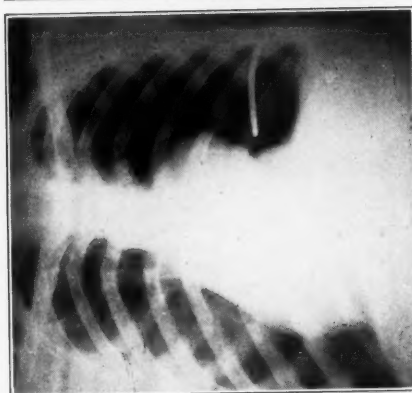


Fig. 2. The same chest as in Fig. 1, after withdrawal of fluid.



Fig. 1. Hydropneumothorax, right side.

The case that I am reporting is as follows: H. H., age 20, with a history of hoarseness for nine weeks and a severe chest condition with a cough. His home physician reported that he had pneumonia. My findings were: complete loss of movement of the left side of the larynx. The X-ray study of the chest showed a right sided hydropneumothorax with the trachea carried well over to the left. After drawing the fluid from the right chest the condition in the larynx gradually went back to normal.

PHYSICIANS' AND SURGEONS' BLDG.

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BENIGN NEOPLASMS OF THE ESOPHAGUS: REPORT
OF A CASE OF MYXOFIBROMA.

ELLEN J. PATTERSON, M. D.,

PITTSBURGH.

Repeatedly one finds in the literature a reiteration of the statement, benign neoplasms of the esophagus are extremely rare affections, there are few cases on record and they deserve only brief mention.

Abraud,¹ Paris, 1911, says: "One knows relatively little of benign tumors of the esophagus. I have been obliged to make a veritable round of the cities to gather up twenty cases. They exist, no doubt. They are, you understand, very interesting not only because of their variety but on account of the favorable prognosis which is attached to their discovery."

In 1926, Vinson² stated that in approximately 4,000 patients complaining of dysphagia examined in the Mayo Clinic, benign tumor was found in only three cases.

In Chevalier Jackson's³ last publication on bronchoscopy, in 1927, he dismissed the subject in a brief chapter of seventeen lines, stating that true benign tumors of the esophagus are rare affections.

A thorough, though possibly not an exhaustive search of the literature, with especial care to elimination of all recounts, goes to prove that the above statements are true today in spite of the development of endoscopy. Then the question necessarily arises in one's mind, Are these neoplasms so rare or are we not having the opportunity to observe the cases on account of the absence of symptoms? There is, however, another solution that the cases may have been diagnosed but not put on record.

A yield of 61 cases of benign neoplasm of the esophagus in the period from 1717 to 1932 is a remarkably small percentage, considering the thousands of esophagoscopies which have been performed by the members of this society alone. Before the advent

of endoscopy, diagnosis was made on regurgitation of the neoplasm into the oral cavity or from autopsical findings, and many of the latter were discovered during routine postmortem, the patient having evinced no symptoms referable to the esophagus during life.

Of the 61 cases garnered from the literature, only 42 are reported with any details, and some of these are meager. Others are reported only by the name of the surgeon and type of growth.

Benign neoplasms of the esophagus may be adenoma, aberrant thyroid, cysts, fibroma, hemangioma, leiomyoma, lipoma, lipomyoma, myoma, papilloma or polyps.

TYPES OF BENIGN NEOPLASMS OF THE ESOPHAGUS.

Aberrant thyroid.....	1
Adenoma.....	2
Dermoid cyst.....	1
Epithelial cysts.....	3
Fibroma.....	2
Hemangioma.....	1
Leiomyoma.....	6
Lipoma.....	2
Lipomyoma.....	1
Myoma.....	8
Papilloma.....	8
Polyps.....	26
	—
	61

Tabulation of the types of benign neoplasms of the esophagus indicates that polyps are the most common benign growths found with myoma and papilloma next in frequency.

Making deductions from the findings recorded in the 42 cases reported with details, we determine that benign neoplasms of the esophagus are more frequently single than multiple and vary in size from a current to one elongated so that it extended from the introitus to the cardia.

They are practically always pedunculated, the pedicle being more frequently long than short, allowing of great mobility. The pedicle is thin, firm and fibrous with a marked degree of elasticity. It may be so long as to permit regurgitation of the growth until it projects beyond the teeth.

In appearance they may be soft or firm; smooth, irregularly lobulated or covered with papillae, and from their point of attachment hang down more or less in the esophagus and at times stretch it to a considerable extent. They may be pear-shaped or elongated and one is described as having four fangs with a single root and pedicle.

The smallest recorded is the size of a currant and the largest 22.5 cm. in length, reported by Vinson,⁴ in 1922, which extended 11.5 cm. beyond the teeth.

Stewart⁵ reports two cases of myoma associated with diverticulum of the esophagus where the neoplasm impinged on the orifice of the diverticulum.

LOCATION OF NEOPLASM IN ESOPHAGUS.

UPPER THIRD		MIDDLE THIRD		LOWER THIRD	
Aberrant thyroid	1	Fibroma	1	Adenoma	1
Adenoma	1	Hemangioma	1	Epithelial cyst	1
Lipoma	2	Leiomyoma	4	Fibroma	1
Myoma	2	Papilloma	1	Leiomyoma	2
Papilloma	1	Polyps	2	Myoma	4
Polyps	7	—	—	Papilloma	3
—	—	9	—	Polyps	3
14	—	—	—	—	15

A consideration of the location of benign neoplasms of the esophagus shows that the largest percentage occurs in the lower third and the smallest number in the middle third, though they are fairly evenly distributed throughout the esophagus. Polyps and papilloma are the only types found in all parts of the esophagus.

ETIOLOGY.

Benign tumors are ordinarily superficial, implanted in the mucosa; however, they may be implanted under the mucosa and may even originate in the musculature (myomata). They do not infiltrate, hence are not malignant but many benign growths have a strong tendency to recur and may recur at the site of a scar, causing a seeming induration and erroneous diagnosis.

The etiology of polypoid tumors is obscure. Morell Mackenzie,⁶ in 1884, suggests that they probably originate in most cases

in chronic inflammation and, assuming analogy of polypi in the nose, one would presume that the inflammation in question should be found in the structure subjacent to the pedicle, but one does not find inflammation in the tissues underlying the pedicle.

Most benign tumors are polypoid on account of the dragging action of peristalsis. They may be due to the passage of food through a narrow portion of the pharynx, where the transverse folds of the mucous membrane are but feebly developed. When such a fold is pushed out of place it can be more stretched and irritated by constantly recurring distention, thus becoming the starting point of the growth. And the pedicle, from being a few lines in thickness, swells to a considerable size and hangs down more or less in the esophagus. The length of the pedicle may be due to the influence exercised by the bolus of food or is due to contraction of the esophagus which draws on it.

Papilloma are really papillary fibroma. They may be rich in blood vessels and therefore tend to bleed readily.

Lipomas occur frequently but are found chiefly at autopsy as small rounded elevations in the submucosa. It is possible for these tumors to become pedunculated.

Leiomyoma occur chiefly in pre-existing smooth muscle tissue and are rarely seen in the esophagus. Coates⁷ noted in leiomyoma the relative absence of fibrous tissue, as compared with uterine fibroma, but considers that that corresponds with the amount of fibrous tissue normally present in the uterus compared with the muscular coat of the alimentary tract.

SYMPTOMS.

Benign neoplasms of the esophagus rarely give rise to symptoms of any degree of severity and some cases are asymptomatic. Sometimes the symptoms are so slight that one doubts the presence of a growth unless it becomes large enough to cause difficulty in swallowing.

The symptoms of all neoplasms of the esophagus are practically the same; therefore one cannot make a diagnosis from symptoms but as compared with cancer, dysphagia, as a rule, progresses much more slowly and it may be years before it gives rise to serious interference with swallowing.

Benign neoplasms, when large enough, may produce stenosis, with extreme dysphagia, nausea, regurgitation of food and marked dilatation of the esophagus, and in some cases may cause spasm of the esophagus, usually intermittent.

Neoplasms with a very long pedicle may be regurgitated into the oral cavity or into the larynx, causing hoarseness, paroxysmal cough and intermittent stenosis of the larynx with dyspnea.

DIAGNOSIS.

Diagnosis is made primarily by the mobility of the neoplasm and its being pedunculated, and unless it is regurgitated into the oral cavity the only means of making a diagnosis is by esophagoscopy.

Dyke⁸ notes that diagnosis of small benign neoplasms is readily perceived by means of the esophagoscope, but large, elongated neoplasms, originating in the upper third of the esophagus, extending to the cardia, with dilatation of the esophagus, would not be readily diagnosed. If the pedicle were seen diagnosis might be made, but once this was passed there would be nothing to indicate that what was actually the surface of the polyp was not the wall of the esophagus.

Sommer,⁹ in 1923, reported the only case in which a diagnosis had been made by radiology. In this case the tumor was lobulated and the diagnosis was made from the fact that streams of barium could be seen passing in different directions along the clefts between the lobules.

Pape and Spitznagel,¹⁰ 1931, reported that roentgenologic diagnosis of myomas of the esophagus is possible. The smaller myomas differ from carcinoma by the absence of infiltration of the wall and the smooth contour of the mucosa. They are differentiated from mediastinal lymph glands, leading to compression phenomena by the fact that they are close to the mucosa.

Large polypoid myoma can be recognized by the fact that the contrast media flows around them, the continuous band of the contrast shadow being split, while it stays constant and continuous in the case of substernal goiter which might displace it entirely. Carcinoma usually shows only one fixed narrow canal.

There is no absolute means of making a diagnosis between benign and malignant neoplasm except esophagoscopy, unless the growth is regurgitated into the oral cavity.

TREATMENT.

The earliest cases reported as treated were Vater's,¹¹ in 1750, where the neoplasm separated spontaneously and was regurgitated, and Munro's,¹² in 1763, where the neoplasm was ligated and later separated and passed with the stool.

In 1818, Dubois¹³ ligated a neoplasm which separated, was regurgitated into the larynx during sleep and the patient died of asphyxia.

Mackenzie,¹⁴ 1874, reports two cases of neoplasms removed with the probang.

Since the advent of esophagoscopy, benign growths of the esophagus have been removed by esophagoscopy without radical removal of the base, as radical removal of the base is usually contraindicated.

CASE REPORT.

Case 1.—J. L. C., age 54 years, was referred to Dr. M. W. Kneidler, with a history of having a growth in his throat for six months which would come up on his tongue. He had had no soreness in the throat until recently, no difficulty in swallowing, no pain or sensitiveness.

On indirect examination of the pharynx a smooth fleshy growth was seen lying on the tongue, which appeared much like an extensive prolongation of the uvula. (Fig. 1.) It was impossible to trace the pedicle. The growth would disappear and by a series of contortions and maneuvers he could regurgitate the growth again into the mouth.

December 22, 1931, at the Allegheny General Hospital, without anesthesia, we introduced a ligature into the proximal end of the growth in order to make traction. Introducing the esophageal speculum and following the pedicle to its attachment it was found on the left side of the esophagus about 2 cm. below its upper border. The pedicle was very tough, had great elasticity and pulled the esophageal wall into the speculum. The pedicle was so

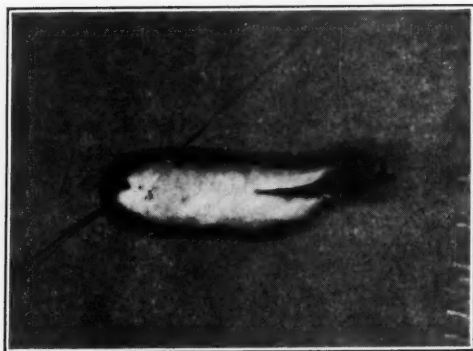


Fig. 1. Growth removed from upper end of esophagus.

tough it was difficult to sever with forceps and tore during removal. The growth was insensitive, and the patient expectorated not more than a teaspoonful of blood. There was no reaction, no bleeding, and six weeks after removal the throat appeared normal.

MICROSCOPIC REPORT.

Dr. Samuel R. Haythorn reported that the specimen consisted of a tumor mass that measured 7 by 1.5 by 1.1 cm., and at the proximal end it measured .5 cm. in thickness. The surface was smooth, slimy and pinkish red in color. It was firm in consistency throughout and homogeneous.

The section of tumor (Fig. 2) was covered with squamous epithelium. The stroma consisted of rather loose fibrous connective tissue in which there were many thick-walled blood vessels and many capillaries. The vessels were surrounded by whirls of fibrous connective tissue which showed a proliferation of fibroblasts. The structure showed edema. The cells were stellate (Fig. 3), and the intercellular substance was mucoid. Diagnosis: Myxofibroma.

Dr. Haythorn further stated that the differential point between myxoma and edematous fibroma is the presence of large stellate

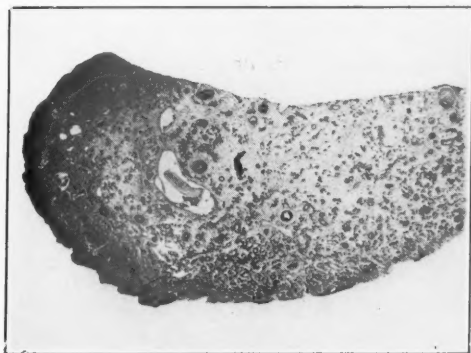


Fig. 2. Section of myxofibroma removed from esophagus.

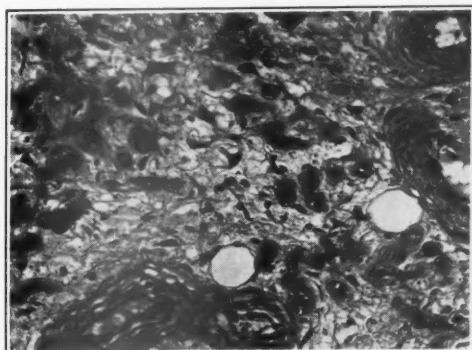


Fig. 3. High power magnification of section of myxofibroma.

cells in place of typical fibroblasts. Some pathologists state that the stellate appearance is due to a large amount of edema in a growth which permits the fibroblasts to swell and assume a stellate appearance. In this case the interpretation would be that of an edematous fibroma. The growth under consideration is not to be confused with the inflammatory polyps found in the nose and esophagus.

CONCLUSIONS.

1. Benign neoplasms may occur in any part of the esophagus and are frequently asymptomatic, therefore unsuspected.
2. They are practically always pedunculated, allowing of great mobility.
3. Conclusive differential diagnosis between benign and malignant neoplasms can be made only by esophagoscopy.

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LXXVI.

CICATRICAL STENOSIS OF THE ESOPHAGUS: CASE REPORT ILLUSTRATED WITH LANTERN SLIDES AND MOTION PICTURE.

E. G. GILL, M. D.,

ROANOKE, VA.

State and Federal legislation requiring manufacturers to properly label caustic solutions and powders has unquestionably lessened the incidence of strictures of the esophagus, but much missionary work is yet to be done before this condition is brought under control. In a review of the literature, one finds that about one case in every fifteen of esophageal disease is stenosis from caustic alkali burns.

AGE OF PATIENTS.

The greatest number of lye burns occur in children. The strictures are more likely to be multiple than single. The strictures are usually located at the levels of the anatomic or physiologic narrowings of the esophagus. Mosher mentions the frequency of cicatricial stricture at the beginning of the liver tunnel.

DIAGNOSIS.

The diagnosis is not difficult if one will carefully consider the history, complete physical examination, roentgen ray and fluoroscopy with opaque mixture. Then esophagoscopy if the patient's physical condition permits.

TREATMENT.

In order that we may more fully appreciate the safety and efficiency of the present-day method of treatment, the older methods will be briefly reviewed.

The older treatments varied from an injection of a 15 per cent alcoholic solution of thiosinamin beneath the skin between the scapulae (Telleky, 1902), to applying caustics through an

esophagoscope to the stricture in hopes it would eat its way through—up to the radical operation of transpleural and transperitoneal esophagogastrostomy, and even further by three plastic operations to exclude the thoracic esophagus by antethoracic esophagoplasty, where plastic operations on the thoracic skin and jejunum are done in an effort to make a substitute tube for the cicatrized esophagus.

According to Park's surgery, external esophagotomy was first performed by Goursault, in 1773, with good results. Internal esophagotomy consists of the introduction of instruments into the canal for the purpose of dividing strictures.

Electrolysis has been advocated by Fort and Ginsez. Symonds used a short rubber tube for intubation of the esophagus in incomplete strictures. Abbe's method consisted of using a string saw to cut through a stricture by a combined gastrostomy and cervical esophagostomy, or through the mouth. Billroth used conical shaped rubber bougies filled with shot or quicksilver. Jacobson advocated a tapered stalk of laminaria tied to a string, in the hope that the laminaria could engage in the stricture before it began to swell.

Schede is given the credit of first suggesting retrograde dilatation through a gastrostomy. The feeding of string in an effort to get beyond the stricture was suggested by Dunham and Mixer: with a string guide beyond the stricture, it soon passed through the stomach and could not be extracted.

By pulling the string taut an excellent guide was had over which a bougie could be threaded to follow the course of the string. This method has been strongly emphasized and used extensively by Plummer.

A paper by Dr. W. J. Mayo, in the *Journal of the A. M. A.*, of July, 1899, described an operation devised by Dr. A. J. Ochsner, in which a gastrostomy is done and a silk guide passed either retrograde or from above; to this string is tied a double rubber tube which is pulled up from below and made to engage in the stricture; when the tension on the rubber tube is released it acts as a dilator. As the stricture releases, larger sizes may be used.

The retrograde bouginage described by Gabriel Tucker is a refinement of Ochsner's operation and is by far the safest, sanest and surest method of dealing with this distressing deformity. There is no doubt that with an early gastrostomy, the getting of a string through the esophagus before it closes and gentle retrograde treatment that the vast majority of these cases can be permanently cured.

The literature is rather prevalent with articles on this subject that have been presented to specialists in bronchoscopy and otolaryngologists.

The writer feels that this Society should use its influence in having this subject presented to the medical and surgical sections of our national organizations. There are many physicians in general practice who do not appreciate the importance of early gastrostomy, nor do they understand why it is necessary to treat those patients for a period of years. No doubt all of you have had children brought up to you completely dehydrated and when asked why they did not come sooner, their family physician advised them to wait, as the child might "outgrow it." Every practitioner of medicine, particularly the general practitioner and the pediatrician, should appreciate the importance of the early recognition and the present day method of treatment of this condition.

CASE REPORT.

Patient, aged 17½ months, was admitted to the hospital on April 30, 1928. The parents gave a history of the child having swallowed lye soap four weeks previously. The family immediately consulted their family physician. Nothing was done in the way of medical treatment at this time, as the burn caused by the swallowing of lye was thought to be confined to the mouth and throat. For one week following the accident the child was in a rather profound stupor. For the following ten days she could eat and swallow satisfactorily, but this was soon followed by almost complete inability to swallow. When we saw the child, on April 30, 1928, she was extremely emaciated and could scarcely swallow water. Her weight at that time was about twenty pounds. She weighed ten pounds at birth.

Examination.—We made an esophageal examination on May 2, 1928. The lumen of the esophagus could not be entered, as there was almost complete closure.

We referred her to Dr. J. T. McKinney for X-ray examination. His plates revealed almost complete closure of the entire esophagus. We advised immediate gastrostomy. This operation was done by Dr. W. R. Whitman on May 15, 1928. She remained in the Lewis-Gale Hospital under Dr. Whitman's care for five weeks, during which time she was fed through the gastrostomy wound. She was readmitted to our hospital on June 23, 1928. We immediately begun trying to have the patient swallow a string. At the end of four hours the string was recovered from the stomach through the gastrostomy wound. The dilatation of the esophagus by the retrograde method with the Tucker bougies was begun. This procedure was carried out until October 15, 1930. During the first year of the treatment, the child was fed by the gastrostomy tube, with the exception of a small amount of water which was given by mouth. After the esophagus had been dilated to its normal size the dilatations were discontinued and the gastrostomy wound was kept open for a period of one year. During this period the patient was fed entirely by mouth. At this time we felt that the esophagus would remain patent and no further dilatations would be necessary. The gastrostomy wound was closed by Dr. W. R. Whitman in October, 1931. The child is now able to eat both solid and liquid foods without difficulty. The patient's esophagus was dilated by the retrograde method 356 times. This case is reported in detail, as it represents one of the few cases where we were able to get the full co-operation of the patient's family and the family physician. In the management of this and other cases we made the following observations, which we feel are of definite value in the management of these cases:

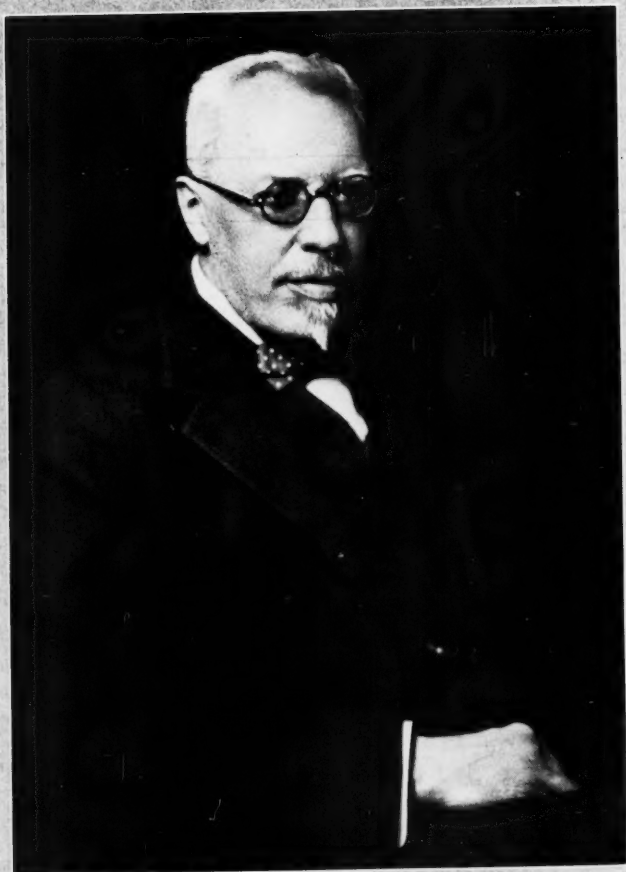
1. Nothing should be given by mouth, other than water, until the No. 20 tube, French scale, goes through readily.
2. After this size tube is reached, we have found that cases improve more rapidly when dilated daily rather than twice a week.
3. The gastrostomy wound should be kept open for at least one year after dilatations have been completed. This is necessary in order to determine if the esophagus will remain patent.

CONCLUSIONS.

1. Gastrostomy is indicated in all cases where patients cannot swallow enough liquids and food to maintain their normal weight.
2. Gastrostomy has a beneficial effect upon the strictured and inflamed esophagus by putting it to rest.
3. Blind bouginage is dangerous and should never be done.
4. The retrograde method combines rapidity of cure with the safest form of dilating cicatricial strictures, and in case the strictures are multiple they are dilated simultaneously.
5. Perforation of the esophagus is impossible when the proper care is used in the selection of the bougie.

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EUGENE ANTHONY CROCKETT

EUGENE ANTHONY CROCKETT.

Eugene Anthony Crockett, former Professor of Otology, Harvard University Medical School, died suddenly on June 13, 1932.

He was born in Calais, Maine, in 1868, and graduated from Harvard Medical School in 1891. He saw military service in the Spanish War, serving first with Battery A, Massachusetts National Guard, and then on the hospital ship "Bay State." He was with the Red Cross in Mexico in 1916, and was commissioned by the Red Cross in Serbia in 1917. Later he became a Major in the American Army attached to the Red Cross in Italy, and in 1918 was decorated by the Italian and Serbian governments.

He served on the aural staff of the Massachusetts Eye and Ear Infirmary from 1891 to the date of his death, as Chief of the Aural Service from 1913 to the date of his resignation in 1924, and as Consulting Aural Surgeon from 1924 to the day of his death.

He was a Fellow of the American Otological Society, and its President in 1925; the American Laryngological, Rhinological and Otological Society; the American Medical Association and the American College of Surgeons. At the time of his death he was Walter A. LeCompte Professor Emeritus of Otology, Harvard University; Consulting Otologist, Peter Bent Brigham Hospital; Cambridge Hospital; Choate Memorial Hospital; and a member of the Committee on Otosclerosis of the American Otological Society.

He published the following articles: "An Acute Syphilitic Affection of the Ear" (1897); "A Case of Operation Upon the Lateral Sinus, with Remarks Upon Eight Unoperated Cases" (1894); "Severe Cases of Thrombosis of the Lateral Sinus, Ligation of the Internal Jugular" (1905); "Thrombosis of the Lateral Sinus—When to Operate: What Type of Operation to Choose" (1910); "Tuberculosis of the Middle Ear and Mastoid" (1900); "When Shall We Remove Tonsils and What Type of Operation Shall We Do" (1911).



A. Bishop Campion

ROY BISHOP CANFIELD.

Roy Bishop Canfield was born at Lake Forest, Ill., July 22, 1874, the second son of Eli Lake and Sarah Maria Bishop Canfield. He attended the public schools of Lake Forest. Coming to Ann Arbor in 1892, he graduated from the Ann Arbor High School in the class of 1893, entered the University of Michigan that fall, taking the combined literary and medical course. He graduated with honors in 1897, receiving his A. B. degree. He was elected a member of Phi Beta Kappa. He received his M. D. degree in 1899.

After graduation Dr. Canfield became associated with Professor Fleming Carrow in the Department of Eye, Ear, Nose and Throat. He resigned this appointment in December to accept an externship at the Massachusetts Eye and Ear Infirmary, Boston. The following August he was made Clinical Assistant. Soon after this he went abroad, following his chosen line of study in the University of Fredrich Wilhelm, Berlin, Germany. He became Chief of Clinic of the Jansensche Klinik and Polyklinik, Berlin, in 1903, Assistant Surgeon of the Manhattan Eye, Ear and Throat Hospital, New York, in 1904. During this year he was also attending laryngologist at the New York City Clinic for Laryngeal Tuberculosis.

In the spring of 1904 the Departments of Ophthalmology and Otolaryngology at Michigan were separated and the latter was tendered Dr. Canfield. With his fine preparation for clinical work, his natural organizing and business ability, he developed a clinic of international reputation and one of the best known in the country. Entering the service as Clinical Professor of Diseases of Ear, Nose and Throat in 1904, he was made Professor of Otolaryngology in 1905 and Otolaryngologist-in-Chief to the University Hospital.

Dr. Canfield was married August 6, 1907, to Leila Marchant Harlow, of Boulder, Colo., a sister of one of Boulder's outstanding physicians. On his return to Ann Arbor he settled down to

still harder and still more constructive work, developing further his submucous resection operation. In the Journal of the American Medical Association, 1908, he published "Submucous Resection of the Lateral Nasal Wall in Chronic Empyema of the Antrum, Ethmoid and Sphenoid," and in the same year he published "The Diagnosis and Treatment of Suppuration of the Labyrinth" in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY. Then came his textbook on "Diseases of the Ear" in mimeographed form, which has been revised from time to time. In 1911 he returned to Europe for further study in Vienna.

When we entered the World War, Dr. Canfield was one of the first to offer his services. General Gorgas called him to Washington. In September, 1917, he was commissioned Commander-Major, Medical Corps, U. S. A., Chief of Ear, Nose and Throat Section, Base Hospital, Camp Custer. Later he was ordered to the Neuro-Surgical School and Rockefeller Institute, New York. He was then appointed Chief of Surgery Service, Base Hospital 76, A. E. F., and later Surgical Consultant, Base Section III. He received his honorable discharge January 11, 1919.

Dr. Canfield was particularly active in scholastic activities. As mentioned before, he was elected to Phi Beta Kappa during his pre-medical training, later to Alpha Omega Alpha. He was a charter member of Phi Rho Sigma medical fraternity.

He was a fellow of the American College of Surgeons, a member of the American Otological Society, the American Laryngological Association, the American Laryngological, Rhinological and Otological Society, the New York Academy of Medicine, the American Medical Association, State and County Societies.

He possessed in the highest degree courage, fortitude, honesty, squareness, fairness, tranquillity, equanimity and a courtesy and kindliness that welded them all together into a substantial whole. Dr. Canfield excelled as a consultant. He was a very present help in time of trouble.

There was no better friend. He died as he lived. Returning from a consultation trip, driving at a high rate of speed as was his custom, passing a truck with a trailer a few miles east of Ann

Arbor at a little after 1 a. m., May 12th, his car for some unknown reason left the road, striking a tree. Death was instantaneous.

Dr. Canfield is survived by his wife, his daughter, Barbara, who was on her way to Europe at the time of his death, and a brother, Arthur Lake Canfield of Somerville, N. J.

—*David Murray Cowie, in the Michigan Alumnus.*

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NOSE.

Natural Orifice of the Maxillary Sinus.

Meyerson, M. C. (New York), Archives of Otolaryngology, May, 1932, page 716.

Myerson found he could enter the normal osteum in 81 per cent of 170 cases, and he found the osteum to lie horizontally in 34 cases, vertically in 98 and obliquely in 6. He did not find proximity of the middle turbinate to the lateral wall to be of any interference in passing his cannula. In the discussion of this paper, normal accessory ostia are mentioned and also the middle meatus puncture as a more desirable method than inferior meatus puncture.

TOBEY.

External Operation on the Frontal Sinus: Causes of Failure.

Anderson, Carl M. (Rochester), Archives of Otolaryngology, May, 1932, page 739.

The author reviews the history of the frontal sinus operations and states that in this series of 161 cases a modification of the Killian and Lynch operation was used. The second stage or external operation was preceded by an intranasal operation. Of the 161 cases, 112 had been previously operated on externally. In a large percentage of these cases failure was due to improper operative procedure or too early interference. He advocates a drainage tube in the nasofrontal duct for some time following the operation.

TOBEY.

Association of Osteomyelitis of the Skull and Nasal Accessory Sinus Disease.

Wilensky, Abraham O. (New York), Archives of Otolaryngology, June, 1932, page 805.

The author writes a very detailed account of osteomyelitis of the skull, and has twelve cases of his own that occurred complicating 306 admissions to the Mt. Sinai Hospital with nasal accessory sinus disease. He reviews the literature very thoroughly. He feels that the fatal infection is usually postoperative when the diploe of the frontal bone is invaded. The usual complications occur, including also lateral or longitudinal sinus thrombosis and

broncho or lobar pneumonia. He believes in very extensive operating once the osteomyelitis becomes evident and feels that granulations of the wound and continued high temperature suggest sequestrums. His mortality is high but not stated. TOBEY.

LARYNX.

The Larynx—A Site of Infection in Certain Diseases of the Lymphatic Glands: Demonstration of Specimens.

Jobson Horne, M. D. Proceedings, Royal Society of Medicine, reported in the Journal of Laryngology and Otology, December, 1931, Vol. XLVI, No. 12, page 849.

The author refers to a paper he read in December, 1901, and published in the *Journal of Laryngology and Otology*, Vol. XVI, page 684, in which postmortem findings in a group of cases which presented the clinical features of Hodgkin's disease. This term is used in a comprehensive sense, to cover the various terms used in literature, such as adenia, lymphadenia, lymphadenoma, lymphadenosis, lymphogenic diathesis, lymphoma, lymphomatosis, lymphosarcoma, malignant lymphoma, pseudoleukemia, a nomenclature in itself evidence of uncertainty.

The cases had two important points in common. Firstly, ulceration of the larynx—the ulceration being situated on the glottic aspect of the arytenoid region and the posterior part of the vocal cord, and secondly, the enlargement of the lymphatic glands in the immediate neighborhood.

The postmortem showed general enlargement of lymphatic glands of the neck, as well as the tracheal, bronchial and anterior and posterior mediastinal glands. Though Horne's paper was published thirty years ago, it seems to have been missed by those who examine only the pharynx. He wished again to point to the larynx as being concerned with Hodgkin's disease.

GOLDSMITH.

An Analysis of 126 Cases of Malignant Disease of the Upper Air Passages Treated During a Period of Ten Years, 1921-1930.

Lionel Colledge and Roydon Peacock (London). The Journal of Laryngology and Otology, Vol. XLVII, No. 111, page 161.

The present status of laryngofissure, lateral pharyngotomy and complete laryngectomy in malignant disease. The series consists of 126 cases. Intrinsic laryngeal, 61 cases; extrinsic, 48 cases;

mesopharynx, 9 cases, and maxillary antrum, 8 cases. The small number of postcricoid growths is due to these having been sent for an opinion only and were mostly treated by gastrostomy.

With laryngofissure Mr. Colledge records a lasting cure in 80 per cent. He thinks a proper selection of cases in the future should raise the percentage to 90. Hospital cases, especially when laryngectomy was carried out, do not do so well as private cases, owing to the less robust physical condition of hospital cases and to the unavoidable changing of postoperative attention.

Eleven cases of laryngofissure had a recurrence of the disease, yet, after a subsequent laryngectomy, seven of those cases remained free from disease.

In the extrinsic cancers a certain number were operated upon by a pharyngectomy or a pharyngo-laryngectomy. Two important points emerge from these copious records, viz., the operative mortality is much heavier in hospital patients than in private practice and the mortality diminishes markedly as the experience and skill of the operator increases. Sepsis causes the most fatalities, but when the wound was left open by uniting the skin edge to the mucous membrane very much better results ensued. Eight cases of malignant disease in the nasopharynx died within two years, seven of those in the first year. The antrum cases were treated by surgery alone or with radium, but in the majority this was ineffective.

In the larynx and lower pharynx, radiation has failed to establish itself, and the first choice must be given to surgery in the hands of experts. In the intrinsic form it has given better results than can be claimed with cancer or any other internal region of the body. Some types of extrinsic manifestation can also be easily cured. With such well established results, practitioners should strive after early diagnosis and will feel justified in advising surgery.

There are at present three patients who possess a larynx perfectly normal in appearance after treatment by radium and who would otherwise have undergone total laryngectomy. The results, however, have been so disastrous in other patients that these three cases seem only to indicate that radium should be reserved for exceptional cases, and that he who undertakes this work

should be prepared to perform any of the great operations on the larynx and lower pharynx or to use radium if there are circumstances which demand its use.

The importance attached to this paper by Colledge is attested to by an editorial in the British Medical Journal, April 2, 1932, under the title of "Cancer of the Larynx." GOLDSMITH.

Laryngeal Vertigo.

Wilson, J. Gordon (Chicago), *Archives of Otolaryngology*, April, 1932, page 534.

In this article, a number of cases of laryngeal vertigo are reviewed and the author reports one case.

Cataplexy is shown to be closely associated. The theories of the causation are discussed (vascular and reflex). The condition is characterized by a sudden fall to the ground and a speedy recovery, with or without loss of consciousness. TOBEY.

Epithelial Polyp of the Larynx Demonstrated by the Roentgen Ray.

W. H. McGehee, M. D. (Cincinnati), *Radiology*, 19:60 (July), 1932.

Considerable interest has been manifest during the last three years in the study of the neck in the lateral projection. Such studies have been stimulated by the original work of Brown and Reineke, in 1928, and later by the excellent monograph of Hayes. Careful review of the literature has failed to reveal any cases of polyp or papilloma of the larynx demonstrated by the roentgen ray, with the exception of the single case in the Hayes publication. This lack of similar cases in the literature and the age of the patient to be presented seem sufficient to justify this case report.

R. M. (Case No. 2968), white, male, age five years, entered the Cincinnati General Hospital complaining of loss of voice. The onset of the present illness began with hoarseness ten months before admission. The hoarseness grew progressively worse until the child could not speak above a hoarse whisper. There had been no obstruction to breathing, only occasional choking on swallowing. The past history was irrelevant. General physical examination was essentially negative.

Roentgen study (lateral view of the neck) showed a tumor mass, approximately 0.5 cm. in diameter, arising from the larynx at the level of the fourth cervical vertebra. The laryngeal ven-

tricle was not demonstrated. It was assumed that the tumor was encroaching on the vocal cords. Impression: Polyp of the larynx.

Direct laryngoscopic examination disclosed a growth about the size of a pea, situated in the anterior commissure, hanging between the vocal cords. The cords were reddened and thickened.

At operation the clinical and roentgen findings were confirmed. The voice was definitely improved when the child was discharged.

PROETZ.

EAR.

Otogenic Basilar Meningitis Without Symptoms (*Meningite basillare otogena asintomatica ecc*).

E. Rubattelli (Padua), Valsalva, 8:99 (Feb.), 1932.

From the clinic of Professor Arslan, fourteen cases of basilar meningitis complicating acute or chronic otitis are reported, in which meningeal lesions out of all proportion to the symptoms were discovered on operation or postmortem. Symptoms include deep, constant frontal headache, pale skin, slowed pulse, changed disposition, slightly increased spinal fluid pressure, although the fluid is generally clear; negative general nervous system, cranial nerves and eye-grounds. Spinal fluid chemical and physical properties change late, when bacteria may be demonstrated. Serofibrinous exudates, often frankly purulent, cover the whole base of the brain, often extending up the frontal and temporal lobes without any localizing signs. Several germs may be found in such exudates, tallying with those noted in the ear discharges. Treatment was limited to extensive mastoidectomy (radical in the chronic cases) with dural exposure. Nine only of fourteen cases were operable and only two survived.

FENTON.

Surgical Treatment of Otosclerosis (*Sur le traitement chirurgical de l'otospongiose*).

Prof. M. Sourdille (Nantes), Rev. de L. O. R., 53:281 (March), 1932.

As previously reviewed in the ANNALS, Prof. Sourdille is continuing his operations on the ossicles and external semicircular canals on the following indications: deafness must show negative Rinne and prolonged Schwabach, with bone conduction for 128 d. v. fork of from 20 to 30 seconds; also, the tympanum must be clean, with intact drum and incus; results are better if the eustachian tube is still permeable. Time of operation: when the ossicu-

lar system can scarcely be set into vibration—that is, perception of the whispered voice at 20 inches or less.

Results make resumption of a profession possible to the half-deafened; to those previously almost totally deaf, they have permitted the use of electric aids and thus auditory re-education, including training in lip reading. Sourdille admits the extreme difficulty and delicacy of his procedures, which are carefully illustrated.

FENTON.

Deep Osteitis of the Petrosa (L'ostéite profonde du rocher).

J. Ramadier (Paris), Ann d'Otolar., 1:1300 (Dec.), 1931.

The author reminds us of Italian and French communications on this subject dating from 1903 to 1914, but considers recent work of Eagleton and Kopetzky to be in advance of current European practice. He follows the indications and technic of Eagleton, which are admirably illustrated by drawings showing the unlocking of the tegmen and removal of the squama and zygomatic root. Three successful cases are reported.

FENTON.

Scalp Tenderness as a Sign of Meningeal Irritation in Acute and Chronic Mastoiditis (La dolenzia del cuoio capelluto quale segno d'irritazione meningeae ecc).

Prof. C. Borri (Bergamo), Boll. delle Mal. dell Orecch. Go'a, Naso, 50:1 (Jan.), 1932.

Explaining Jason Dixon's observations along this line, Borri considers that the dura should be exposed above the mastoid in all such cases; the pain comes from dural fibers issuing mainly from the middle meningeal nerve and is also noted in traumatic lesions of the middle fossa.

FENTON.

Treatment of Mastoiditis With X-Rays.

William L. Ross, M. D. (Omaha), Radiology, 18:1124 (June), 1932.

The author reports forty-one cases of mastoiditis treated by X-ray. Sixteen acute cases were seen in children. Of the adults, fifteen were acute, seven subacute and three chronic. Ten of these patients had been previously diagnosed acute mastoiditis by others and operations had been advised.

The diagnosis was based upon the character and location of the pain, the degree of tenderness to superficial or deep pressure and the quantity rather than the character of the discharge from the ear. Estimate of bone destruction was based upon a comparison of the diseased side with the normal.

The technic, number of treatments and interval were varied to suit the cases. In the acute cases, roentgen treatments were given every eight to twelve hours until the pain subsided, which usually occurred after three to five treatments. Relief of tenderness required five to seven more. The discharge usually increased in quantity for from two to four days, when it began to lessen rapidly. No mention is made of complete cessation of discharge.

The advantages claimed are the shortened course of the disease and the thorough covering of the diseased area. The disadvantages are temporary loss of hair over the area exposed to X-ray. The hair returns in about three months. There is also some dermatitis of the exposed area, said to be controllable "by proper filtration, the application of salted butter once a day over the exposed area, and the application of a very mild high frequency current."

The case histories appended to this report leave much to be desired, being notably deficient in detail. Apparently no bacteriologic observations were made and in only a few are there reports of blood cytology. The diagnosis was frequently made on mastoid pain and cloudiness to X-ray. The description of previous middle ear infection is too vague to be convincing. The symptoms in some cases may well have been produced by periosteal abscess or an otitis externa.

The first conclusion that X-ray treatment is applicable in almost all phases of mastoiditis seems untenable in the light of the evidence offered. If this treatment has any merit, it deserves more scientific study and analysis.

PROETZ.

The Use of Periosteal Flap Grafts in Mastoid Operations.

O. Popper (London). *Journal of Laryngology and Otology*, February, 1932, Vol. XLVII, No. 2, page 126.

The author, after an experience of twenty-three cases in which he used a periosteal graft from the skull to line the mastoid cavity, has no hesitancy in stating that it far surpasses skin or muscle.

The suitability of a periosteal graft is apparent when one considers the following points:

1. It has a broad attachment to the parent tissue and is adjacent to the operative field.
2. It has its own blood supply.

3. It will flush the cavity continually with serum rich in antibodies.

4. It is more viable, being a less highly specialized tissue than muscle, fat or skin.

5. It does not obliterate but merely lines the cavity.

6. The aditus ad antrum remains free and unobstructed for drainage.

7. It is thin and easily manageable.

8. It can be mobilized in any direction without torsion.

9. Lastly, and most important, it is the natural covering of bone.

Technic: The procedure hardly deserves the title of technic. The usual postaural incision is made, through skin and subcutaneous tissue only. The periosteum is now exposed in a forward direction to the auricular fold. The posterior skin flap is now retracted and dissected off the periosteum as far back as possible. Occasionally, the posterior skin flap is split at about the middle for about one-half inch to secure easier exposure, the object being to obtain a large piece of periosteum—about two square inches. A horseshoe-shaped incision is now made through the periosteum, the convexity facing backwards and being about one and one-half inches behind the auricular fold. The open part of the horseshoe corresponds to the fold of the ear. The periosteum is carefully raised from behind forward to its attachment in the fold.

The osteal operation is now performed.

The rationale of the periosteal flap procedure is to cover the wounded bone. The free edge of the periosteum is secured with two pair of artery forceps. The portion between the forceps and the auricular fold is now pushed gently into the cavity so that its osteal surface becomes closely applied to the walls of the cavity, to which it is to become firmly adherent. The flap is pressed on to the facial ridge if the bridge has been taken down.

When the outer attic wall has been removed or the Jenkins operation performed, a narrow strip of periosteum is pushed well into the epitympanum. This strip can either be freshly prepared or cut off from the original flap by an incision parallel to the upper edge and about one-fourth inch in width and one and one-

half inches long. It remains attached in the auricular fold, of course.

The advantages of this method are:

1. The minimum of bare bone remains exposed after operation.
2. One uses to cover it a tissue which is fashioned by nature to do so.
3. Healing is more rapid, owing to profuse blood supply.
4. The liability to reinfection (the cause of chronic otorrhea) is very materially reduced.
5. The meatus is preserved, a factor not generally recognized as of importance in the preservation of hearing.
6. The cosmetic result is very good.
7. It requires very little additional time or trouble.

GOLDSMITH.

Otosclerosis—A Clinical Analysis.

Dan McKenzie (London). *Journal of Laryngology and Otology*, February, 1932, Vol. XLVII, No. 2, page 92.

The author has analyzed over 600 private cases of deafness and has carried out a very valuable piece of clinical research. An abstract, curtailed as it is bound to be, cannot do justice to the paper.

He has divided his cases into three classes:

1. Primary otosclerosis, total 411 cases; males, 130 (31 per cent); females, 281 (69 per cent).
2. Secondary otosclerosis, total 129 cases; males, 52 (40 per cent); females, 77 (60 per cent).
3. Incipient otosclerosis, total 82 cases; males, 32 (39 per cent); females, 50 (61 per cent).

Primary otosclerosis, the largest division, is made up of cases in which typical deafness is present without any evidence of catarrhal or suppurative disease in the ear. In order to make sure that definite otosclerosis was being considered, he rejected for this division and from the secondary division also, all cases that did not manifest well marked deafness, the rule being inability to hear the spoken word at a distance of more than three feet.

Secondary Otosclerosis—Here were placed cases of progressive obstructive deafness supervening upon suppuration of the middle ear (active or quiescent), and upon manifest catarrh of the middle

ear without meatal discharge. As both of these conditions cause deafness, the author considered only those in which the deafness was progressive and cochlear involvement absent.

Incipient Otosclerosis—Under this classification is placed trifling deafness of long duration, uninfluenced by treatment and not associated with suppuration and catarrh.

This paper stresses the importance of the progressive character of the deafness in otosclerosis. It is also pointed out that the progress may be very irregular. The disease may start at any age, even in the very young, but for a long time remain dormant. Symptoms may not show themselves but in a minor degree, though the disease has been slowly progressing for years. Again very rapid progress may take place. Unilateral otosclerotic deafness is not at all unusual; 67 cases were found in the primary class and 22 in the secondary. As a rule, the disease ultimately becomes bilateral.

GOLDSMITH.

Some Postoperative Results of the Radical Mastoid Operation in Children. Postoperative Otorrhea.

N. Asherson (London). *Journal of Laryngology and Otology*, May, 1932, Vol. XLVII, No. 5, page 317.

The results in 100 consecutive cases of the radical and conservative mastoid operation in children are tabulated. The residual hearing is recorded as is also the results in a few cases in which muscle graft has been used.

These cases occurred amongst a consecutive series of about 1,000 cases of chronic suppurative otorrhea in children up to 16 years of age, 16 per cent of all these cases of chronic otorrhea required operation.

In discussing the mucus secretion from the tympanum following operation, the author makes the following observations: If the tympanic mucosa has been left or regenerated it is a secreting surface. In the normal ear this mucus finds its way into the eustachian tubes and then to the nasopharynx. In the postoperative case the mucus collects, and unless removed stagnates and ultimately becomes purulent. Such an ear is not in the ordinary sense a discharging ear and requires only routine cleansing. One might speak of it as physiologic otorrhea when the mastoid part

is healed, the tympanic cavity is pale and not red and the discharge is mucoid.

The relation of the nasopharyngeal infection to otorrhea is thoroughly discussed. The failure to cure chronic middle ear suppuration by removal of tonsils and adenoids is stressed. It is only in cases of old discharge with a central perforation that the question of tonsil and adenoids should ever arise from the ear point of view. In pure tympanic sepsis a central perforation exists and there is no involvement of the annulus tympanicus. About 50 per cent of all cases of chronic otorrhea in children fall into this group, and 95 per cent can be cured by a single ten-minute ionization with zinc sulphate. Tonsils and adenoids and nasal discharge in this group require attention. Forty per cent of all cases of chronic otorrhea in children have a marginal perforation with granulation indicative of bone disease. Here ionization is no use, but half the cases can be cured by the use of iodine-boric acid powder.

Nasal and pharyngeal operation on this last type of cases simply brings such operations into disrepute. If the pharyngeal symptoms *per se* call for operation, well and good, but it cannot be expected to cure a local focus of bone disease in the tympanum.

Attention to the following points is emphasized in discussing the various methods of local treatment.

1. In a purely tympanic infection, the mastoid being healed, with no granulations in the tympanum, ionization with zinc sulphate will effect a rapid cure.

Attention to the nasopharynx is essential. Thus adenoids, septic tonsils and sinusitis require attention. This is an essential part of the treatment.

When granulations occur in the tympanum, cauterize with 50 per cent silver nitrate, and after a few days 1 per cent iodine powder should be insufflated regularly.

2. Hypertrophied granulations or polypi require removal by scraping, snaring or curetting. Cauterize the base with silver nitrate.

This applies to the mastoid part of the excavation also. If there is much bone disease still present or any atresia of the

meatus present, further treatment by operation is indicated and the infective bone disease should be eradicated.

Amongst the accessory methods to be attempted in very resistant cases are heliotherapy, ionization with very weak solution of silver nitrate or, after thorough cleansing, packing the cavity with strips of gauze impregnated with "bipp."

The discharge persisting after the radical mastoid operation is usually a very chronic one and reaction to treatment disappointing in a large majority of cases, and further operation is usually called for unless the site of origin is localized to one or two definite spots.

GOLDSMITH.

Suppuration of the Petrous Pyramid.

Lillie, Harold L., and Williams, Henry L. (Rochester), *Archives of Otolaryngology*, May, 1932, page 692.

The authors report two cured cases of petrousitis operated on by the Kopetsky approach anterior to the cochlea and above the carotid artery. They call attention to the fact that pneumatization of the anterior labyrinthine group or posterior group of cells makes much more room for operating by one of these approaches.

TOBEY.

Suppurative Meningitis of Otitic and Nasal Origin: Its Relation to Blood Stream Invasion of the Pial Vessels.

Eagleton, Wells P. (Newark), *Archives of Otolaryngology*, June, 1932, page 885.

Eagleton divides otogenic and nasal infection meningitis into (1) subarachnoid space meningitis and (2) arachnoid vessel meningitis. He stresses many localizing symptoms as well as signs for involvement of the various cisterns, and states that No. 1 is curable surgically and No. 2 is fatal. He believes Pneu. III meningitis secondary to pial vessel infection to be of sphenoidal origin, and frequently the mastoiditis is complicated by a silent sphenoid, even in hemolytic streptococcus septicemias.

TOBEY.

MISCELLANEOUS.

The Otolaryngologica Slavica—Volume 4, 1932.

This volume principally presents the report of the first Congress in Otolaryngology of the Southern Slavic countries, which was held in Zagreb, Jugo-Slavia. The occasion of the Congress was the tenth anniversary of the Clinic of Zagreb in Otolaryngology.

In the main, the contents are on the tonsils, dealing with experimental and research work in physiology, anatomy, bacteriology, pathology and therapy, both surgical, semisurgical and non-surgical.

The following thirteen subjects are represented by clinicians from the various parts of the Southern Slavic Countries and are published in English and German. Four of these papers are published *in extenso*.

1. Dr. Vulovic of Beograd—"The Tonsil Problem."
2. Dr. Saltykow of Zagreb—"Pathologic Anatomy of Tonsillitis."
3. Dr. Hercog of Zagreb—"The Tonsil Problem in Regard to Internal Medicine."
4. Drs. Wiskovsky and Fabry of Bratislava—"The Causes and Diagnostic Value of the Inequality in Size of the Faucial Tonsils."
5. Dr. J. Hybasek of Brno—"Sepsis Following Tonsillectomy."
6. Dr. Alcalay of Beograd—"Histologic Examination of Tonsils Relative to the Hemorrhages in Tonsillectomy and Tonsilotomy."
7. Drs. E. Prasek and A. Zuk of Zagreb—"Bactericidal Substances Within the Tonsils."
8. Dr. F. Mihaljevic of Zagreb—"Anginas with an Atypical Blood Picture."
9. Dr. S. Podvince of Zagreb—"The Place of the Faucial Tonsil in the General Lymphatic Apparatus."
10. Dr. N. Jerkovic of Zagreb—"The Effect of the Tonsil Extract on the Leucocytic Count."
11. Dr. B. Chorazycki of Warszawa—"Anatomic Configuration of the Tonsil in Man."
12. Dr. Podvinec of Zaquele—"The Tonsil Problem."
13. Dr. Pejic of Beograd—"Experimental Investigation on the Functions of the Tonsil."

Taking it all in all, these articles or presentations by the above authors are very brief but to the point. Their attitude in regard to the relation of tonsils to general diseases is still very far from the firm conviction and belief that we have in this country. The reason for this can be explained because of the much smaller

number of tonsillectomized patients as compared to the tremendous accumulated material in the United States.

Their research work on the use of the extracts of the tonsillar tissue on the body is very interesting and bears watching for future experimental results.

General sepsis following tonsillectomy is very well described by Hybasek in a case that had an acute lacunar tonsillitis just before operation. The course and involvement of the veins of the neck and their subsequent ligation and resection giving permanent benefit was most interestedly observed by the author and tabulated blood examination in regard to the septic infection were most excellently described.

The paper on the place of the Faucial Tonsil in General Lymphatic Apparatus is a physiologic discussion. It deals particularly with regard to the formation of lymphoid cells and their entrance into the general lymphatic circulation, not necessarily acting as phagocytes. The old chemotactic theory as to the leucocytes is again given, but in a more modern view, and will stand a great deal more scrutiny than in former times. BECK.

The Revue de Laryngologie, Otologie et Rhinologie, published for fifty-three years by Professor Moure of Bordeaux, appears since January of this year in an enlarged format, on excellent paper with many illustrations, under the editorship of Professor Georges Portmann. Announced intentions include rather lengthy leading articles by international experts, and an extensive abstract section. Portmann himself contributes a detailed study of lesions of the auditory nerve in encephalitis to the January and February issues. FENTON.

The Relationship Between Status Lymphaticus, the Thymus and the Suprarenal Glands.

Campbell, Edward H. (Philadelphia). *Archives of Otolaryngology*. April, page 517.

The author reviews the recent literature on status lymphaticus, the thymus gland and the relations of this gland to the suprarenals.

He feels that otolaryngologists should recognize that various degrees of status lymphaticus exist and have an important bear-

ing on the outcome of not only operations on tonsils and adenoids but other operations and diseases in otolaryngology. He feels that we should know the significance of reported thymic enlargements and that it is a symptom of status lymphaticus which in turn is due to deficiency of secretion from the suprarenal glands.

The next step, in his mind, is to learn to detect status lymphaticus and get a suprarenal gland product rich in medullary and cortical elements.

TOBEY.

Relation Between Turbinal Hypertrophy and the Endocrine Sympathetic System (L'ipertrofia dei turbinati nei suoi rapporti col sistema endocrinosimpatico).

A. Pazano (Naples), Arch. Ital. O. R. L., 42:656 (Dec.), 1931.

Predisposing constitutional factors which prejudice the normal balance between sympathetic and parasympathetic impulses and depend often upon glandular deficiencies are to blame for most of these diffuse hypertrophies. Normal vasoconstriction by the sympathetic does not occur, while the parasympathetic vasodilatation is unimpeded. Headaches, attacks of colitis and tinnitus were associated in a number of these cases.

FENTON.

Pins at the Periphery of the Lung: Observations Based on Forty-two Cases.

Jackson, Chevalier, and Jackson, Chevalier Lawrence (Philadelphia), Archives of Otolaryngology, June, 1932, page 861.

Pins tend to lodge head downward in the lungs, and, due to respiratory movements and a catching of the point, are forced further into the lung periphery, and may remain silent for months before the patient begins to have impaired health. Cases are reported and X-ray pictures shown. The authors have found removal of pins from all except the ascending branches of the upper lobes could be done without great difficulty by peroral bronchoscopy.

TOBEY.

Streptococcus Hemolyticus Carriers and Puerperal Sepsis: The Effect of Tonsillectomy on the Incidence of the Organism.

W. R. Logan (Edinburgh). Journal of Laryngology and Otology, Vol. XLVII, No. 3, page 196.

Recent workers have shown that in a large proportion of the more serious cases of puerperal sepsis and in most of those occurring in epidemic form, the streptococcus hemolyticus is the bac-

terial factor. It has been known for some years that this organism is present in the crypts of the tonsils, of a considerable proportion of the population, and an examination of surface swabs gives an underestimate of its prevalence in the throat. It has been found by J. Smith that though the serologic type of streptococcus differed in various cases, the type was the same in the throat of the medical attendants and nurses as in the uterus of the patient in a high proportion of cases.

Various precautionary measures have been instituted, including the wearing of masks by the various attendants. Recently more drastic treatment has been advocated, one of which forms the basis of this paper, viz., the doctors and nurses who are found to carry the hemolytic streptococci in the throat should undergo enucleation of the tonsils. Dr. J. S. Fraser raises the question, "Does tonsillectomy rid the throat of this organism?"

Thirty-eight nurses of the Edinburgh Royal Infirmary were tested. Swabs were taken from the throats of nineteen nurses who had had their tonsils dissected out by Dr. J. S. Fraser, and from nineteen control nurses who had not been subjected to operation. The result may briefly be stated: *Streptococcus hemolyticus* was isolated for 36.8 per cent of the normal control nurses and 26.3 per cent of the nurses whose tonsils had been dissected out. If instead of limiting the bacteriologic investigation to the true hemolytic strains, the pseudohemolytics had also been included the figures would read 73 per cent of normal nurses and 47 per cent of those whose tonsils had been removed.

Dr. Fraser points out that complete removal of the faucial tonsils does not get rid of the lingual tonsils nor the small aggregations of lymphoid tissue on the lateral and posterior walls of the pharynx. This is quite apart from the teeth.

This investigation is very timely, and while it is quite opposed to many reports on the influence of tonsil surgery on the bacteriology of the pharynx, it is hoped that it will lessen the amount of loose talking on the influence of tonsil sepsis in puerperal septicemia.

Note by Dr. J. S. Fraser.—About a year ago I was asked to undertake a tonsillectomy campaign at a maternity hospital. The

idea was that all doctors, nurses and ward maids who were on duty at this hospital should have their tonsils enucleated in order to get rid of the risk of infecting puerperal cases with the streptococcus hemolyticus. My reply was that before undertaking such a campaign I should like to have some proof that tonsillectomy did abolish the streptococcus hemolyticus from the throats of those who had undergone this operation. Accordingly, I appealed to Dr. W. R. Logan to undertake the investigation, the results of which are given in the above paper. GOLDSMITH.

Vincent's Disease.

Professor Leonard S. Dudgeon (London). The Journal of Laryngology and Otology, March, 1932, Vol. XLVII, No. 3, page 188.

Vincent's disease may occur as an inflammatory, suppurative, necrotic, ulcerative, membranous or gangrenous process affecting the gums, mouth, lips, tongue, tonsils, palate or pharynx, associated with slight or considerable glandular enlargement.

Death may occur from gangrene, laryngitis, bronchopneumonia, abscess or gangrene of the lung, or from an intercurrent bacterial infection.

Vincent recognized two forms of affection: (1) Diphtheria-like, showing a firm yellow-white false membrane and only superficial ulceration; (2) an ulcerative type in which the membrane is soft, grayish and foul smelling, and there is ulceration with edema.

The organisms believed to be the cause of this infection of the mouth and throat are *F. fusiformis* and the *Treponema vincenti*. They are found in the crypts of the tonsils, gingival margins, the tartar of the teeth, and in any decayed organic matter in the mouth or around the teeth or gums in children and adults in whom there is no evidence of Vincent's disease.

Etiology.—1. Trauma of any kind. This may be due to extraction of teeth, injection of local anesthetic into the gums, too frequent use of strong chemicals on the mouth and gums, operations on tongue, mouth or tonsils.

2. During the course of, or convalescence from, an acute infection.

3. In cases of chronic disease, especially associated with severe anemia.

4. In wasting diseases.
5. In children or adults, without any apparent reason.
6. Infection from without.

7. Colyer and Sprawson (1931) quote evidence of Clewer that absence from the diet of antiscorbutic vitamins may play its part as a causative factor in Vincent's disease. In 41 cases out of 50 tabulated by Clewer a fresh vegetable ration had not been a part of the diet.

In many cases two or more of the various causes referred to above are acting in conjunction. The frequency of the disease during the Great War was no doubt due to a multiplicity of causes. For these reasons and many others the objectionable term "trench mouth" should never have been introduced.

The chief infections which may be mistaken for Vincent's disease are diphtheria, streptococcal infections and syphilis.

Prevention.—In the prevention of this disease it must be recollected that infection is in most instances derived from the teeth or tonsils. Those who consider that Vincent's disease may be prevented fail to realize that its sudden occurrence without any previous indication of infection is not uncommon. Once, however, the infection is diagnosed, active measures should be adopted to insure that it is under control.

Thorough cleanliness of the mouth is essential, and treatment of the gums with carbolic acid, free chlorin, perborate of soda, iodine or iodine in glycerin, hydrogen peroxide or any other suitable preparation should be employed, and, if necessary, improvement of the patient's general condition and surroundings should be carefully attended to.

Therapeutic.—In mild cases the preventive measures already referred to are the most suitable, and of these freshly prepared chlorin gargle mixed with an equal part of warm water is by far the most valuable local application. The strength of chlorin can be increased if the patient can tolerate it and if it is desirable.

The author is convinced, however, that a too frequent application of powerful "germicides," or even of mild applications, is a mistake, as the only means by which recovery can take place is by repair on the part of the patient's tissues, which should be

given every chance to react. In gangrenous cases, however, the progress of infection may be so rapid that unless vigorous local treatment is carried out, the infection may advance beyond control. Of internal remedies arsenic is, in the author's experience, the most efficient and is of the utmost value, even in cases of gangrene. Glucose should be given internally, as patients may be unable to take sufficient nourishment. Intravenous arsenic should be given to all severe and gangrenous cases and to those who fail to react to the ordinary lines of treatment or in whom relapses occur.

Any concurrent illness or predisposing cause of Vincent's disease should receive adequate treatment at the same time.

GOLDSMITH.

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